"A SIMPLE SLIDE TEST TO ASSESS GRADES OF ERYTHROCYTE AGGREGATION IN ACUTE ST-ELEVATION MYOCARDIAL INFARCTION AND ITS PROGNOSTIC SIGNIFICANCE"

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

In Partial fulfilment of the Regulations
For the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

April – 2015

BONAFIDE CERTIFICATE

This is to certify that **"A SIMPLE SLIDE TEST TO ASSESS GRADES OF ERYTHROCYTE AGGREGATION IN ACUTE ST-ELEVATION MYOCARDIAL INFARCTION AND ITS PROGNOSTIC SIGNIFICANCE"** is a bonafide work performed by **Dr.M.KARTHIKEYAN**, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai - 10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2012 to April 2015.

Prof.Dr.R.SABARATNAVEL,M.D.,
Professor and HOD
Department of Internal Medicine
Kilpauk Medical College
Chennai-10.

Prof.Dr.N.GUNASEKARAN,M.D.,DTCD
The DEAN
Government Kilpauk Medical College
Chennai - 600 010.

DECLARATION

I solemnly declare that this dissertation **"A SIMPLE SLIDE TEST TO ASSESS GRADES OF ERYTHROCYTE AGGREGATION IN ACUTE ST-ELEVATED MYOCARDIAL INFARCTION AND ITS PROGNOSTIC SIGNIFICANCE"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai - 10, under the guidance and supervision of **Prof.Dr.R.SABARATNAVEL, MD,** Professor and Head of the Department of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai

Date: **(Dr. M.KARTHIKEYAN)**

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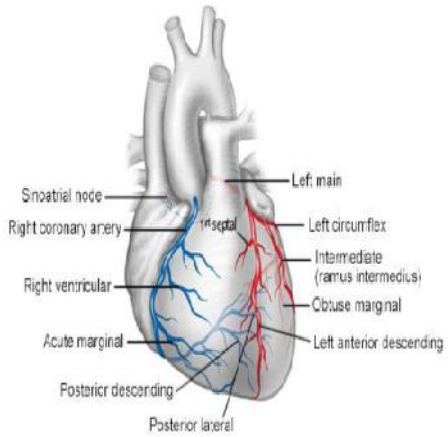

Picture: 3 Blood supply of the Heart

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ABBREVIATION

MI	-	Myocardial Infarction
SCD	-	Sudden CardiacDeath
ECG	-	Electrocardiography
ESR	-	Erythrocytesedimentation rate
LORCA	-	Laser Assisted optical rotation cell analyzer
CFA	-	Cell flow analyzer
EAAT	-	Erythrocyteaggregation/adhesiveness test
ECHO	-	Echocardiography
AMI	-	Acutemyocardialinfarction
CHD	-	Coronaryheart disease
CAD	-	Coronaryarterydisease
HF	-	HeartFailure
PA	-	Photoacoustics
ATS	-	Atherosclerosis
IHD	-	IschemicHeart Disease
CIHD	-	ChronicIHD withCongestiveHeartFailure
LDL	-	LowDensity Lipoprotein
HDL	-	High Density Lipoprotein
VLDL	-	Very Low Density Lipoprotein
CPK	-	Creatininephosphokinase
LDH	-	Lactatedehydrogenase
RBC	-	Red Blood Cells
Px	-	Prognosis
EA	-	Erythrocyteaggregation
STEMI	-	ST elevation myocardial infarction
RV	-	right ventricle
LV	-	left ventricle
AWMI	-	anterior wall MI
IWMI	-	inferior wall MI
RVMI	-	rightventricular MI
RCA	-	right coronary artery
LAD	-	left anterior decending artery
LCX	-	left circumflex artery

CONTENTS

ABSTRACT

Asimple slide testandanalysis of imagewasusedtorevealthe presence ofan acute-phaseresponse insubjectsofacutemyocardial infarction.Erythrocytestendtoaggregatewhenaninflammatory processlike atherosclerosisincoronary artery disease. Evaluationoferythrocyte adhesiveness/aggregation (EAAT)iscurrently availabletothephysiciansindirectly-by erythrocytesedimentationrate(ESR).TheESRcorrelatespoorly witherythrocyte aggregation,hencea simple slide test usingcitratedbloodwasusedtoevaluate erythrocyte aggregation in grades by microscopicallyandalso byusingimage analysis.

AIM OF THE STUDY:

Tostudy erythrocyteaggregation and adhesiveness by asimpleslidetestinsubjects with acuteST-elevatedmyocardial infarction (STEMI) in predicting the outcome within 1week.

METHODS AND MATERIALS:

Fifty patientsofacuteSTEMIwhocametotheICCUofGovernment Royapettah Hospitalwere includedinthe study, onsetofchestpain within 6hrs,Subjectswithretrosternalchestpainpersistingformorethan30 min and with ST segment elevation 1mm in limb leads and 2mm in chest leads in at least two contiguous ECGleadswereincludedinthestudy.CitratedbloodwassubjectedtosimpleslidetestandStainedsmears werereexamined under400X andgraded into fourgrades.

OUTCOMES

To find out whether there is any statistically significance between the grades of erythrocyte aggregation and prognosis of STEMI patients within 1 week.

Good Prognosis: Recovery is good

Bad Prognosis: Patient developed reperfusion arrhythmias, LVF, recurrent Infarction, VT and VF, cardiogenic shock, death from cardiac and non cardiac causes.

RESULTS:

'P' value between the GradesofRBCaggregationandthe prognosisof the patients with STEMI was <0.05.

CONCLUSION:

EAATisa simple bedside slide testfor erythrocyte aggregation,whichindirectly find outthe presenceandproportionof inflammation.Thistest also hasthe potentialto assesstheprognosisofSTEMI patients. Itcanalsobeusedas ascreeningtestfor patients with STEMI to find outhigh-riskindividualssothatnecessary interventions (like PCI)couldbe adopted.

Introduction

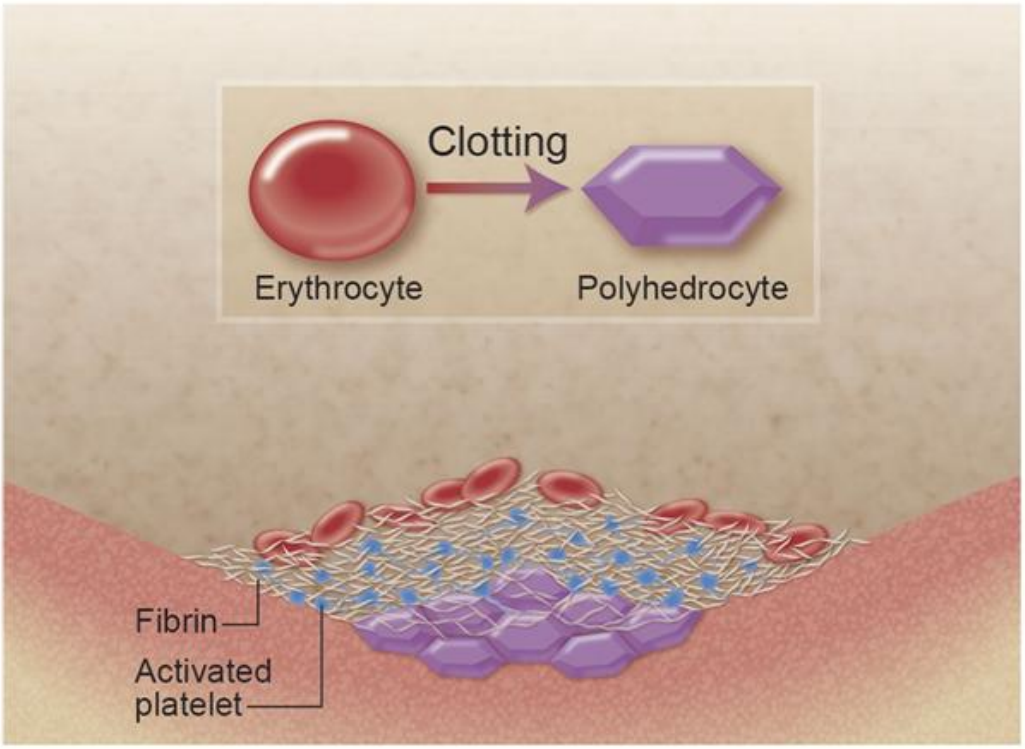
INTRODUCTION

The main cause of death in the world are up to twelve millions death per each year due to cardiac diseases mainly CAD.“MI(myocardial infarction)isthe irreversible necrosisof heartmuscle due toprolongedhypoxia leads to ischemic changes in the cardiac muscle andoccursfromanimbalancebetweenO2supply anddemand,frequentlycaused by atheromaruptured to form small plaques that will leads to thrombus development in a coronary arteries. [1]

Red blood cells are mainly involved in a formation and development of clot orthrombus or clot. Red cells have some adhesive molecules such as proteins. They have more intensity of attachment to the endothelium of coronary blood vessels. EAAT or red cell aggregation test include in acute-phaseresponse that leads toalterations inthe membrane component phospholipid, especially in a subjects with high lipid profile. [2, 3]

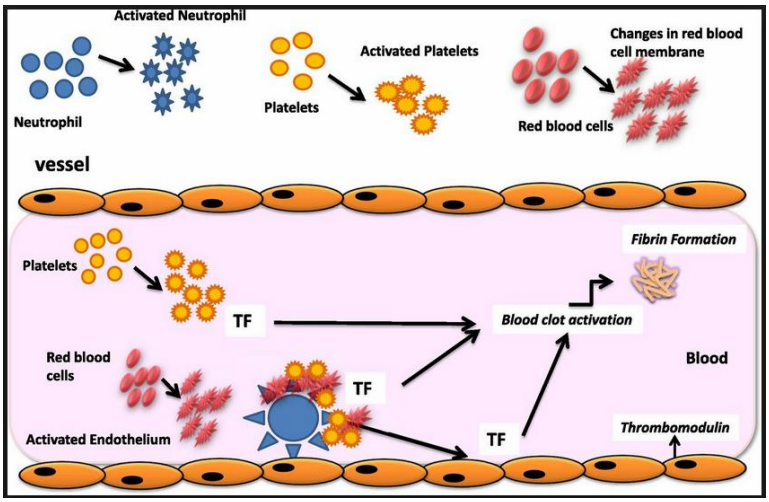
In normal, red cells act against free radicals i.e. reactive O2 or N2 species. Once free radical production more than antioxidant activity of red cells. At of this stage the morphology or shape of the red cell is altered that leads to aggregation or rouleaux formation. Once it is happened, the red cells gets the property to attachment to the coronary

vascular endothelium, this finally leads to the vascular endothelial damage. If damages occur in the vascular endothelium that will leads to adherence of other factors of atheroma formation.[2,4,5,6]



Picture: 1 shows structural changes in RBC tends to aggregation

Red cells aggregation is independent RF for clot or thrombus formation leads to blockage in both arterial system and venous systems. These properties of red cells aggregation, would also be easily identifiable in the small peripheral capillaries with poor circulation. In this study shows, that there is relation between the atheroma formation in the vascular system and red cells aggregation.



Picture: 2 shows red cells leads to thrombus formation

Erythrocyte or red cells aggregation is find out indirectly through Erythrocyte Sedimentation Rate (ESR). But this has not adequately correlates with EA. EA can be observed accurately by INFLAMET (designed in Israel), LORCA, CFA and MyreneRheometer. [2,8,7] These are expensive tests, here using a simple slide test done at bed side and very easy method, low expensive, rapid method for detecting the grades of red cells aggregation by EAAT (EA and adhesiveness test) and directly seen andassessing the grades of EA. This method is usefulfordetect any inflammation occurs in the vascular system.[9]

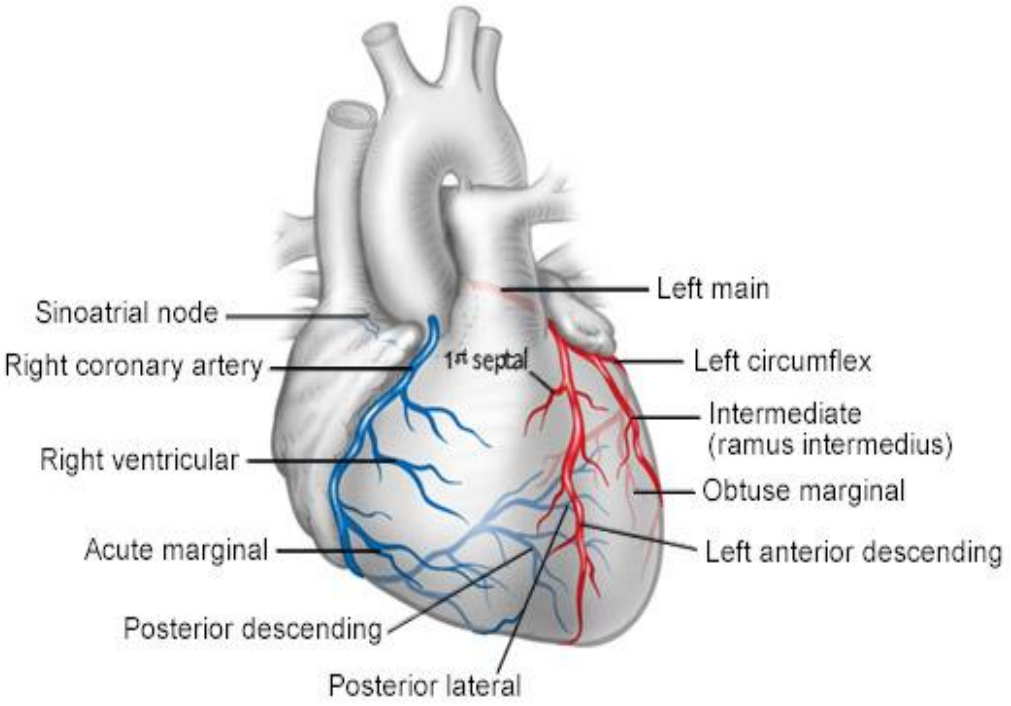
This simple test will be donetohelpthephysician to know about the pathology of ASTEMI (acute ST segment elevation MI) patients with their prognosis and therebyin giving appropriate therapy. Inthis physician subjectively assigning the grades of EA under the microscope (oil immersion field) from subjectsof ASTEMIandcompared with their prognosis.

Literature Review

LITERATURE REVIEW

ANATOMY: BLOOD SUPPLY OF THE HEART

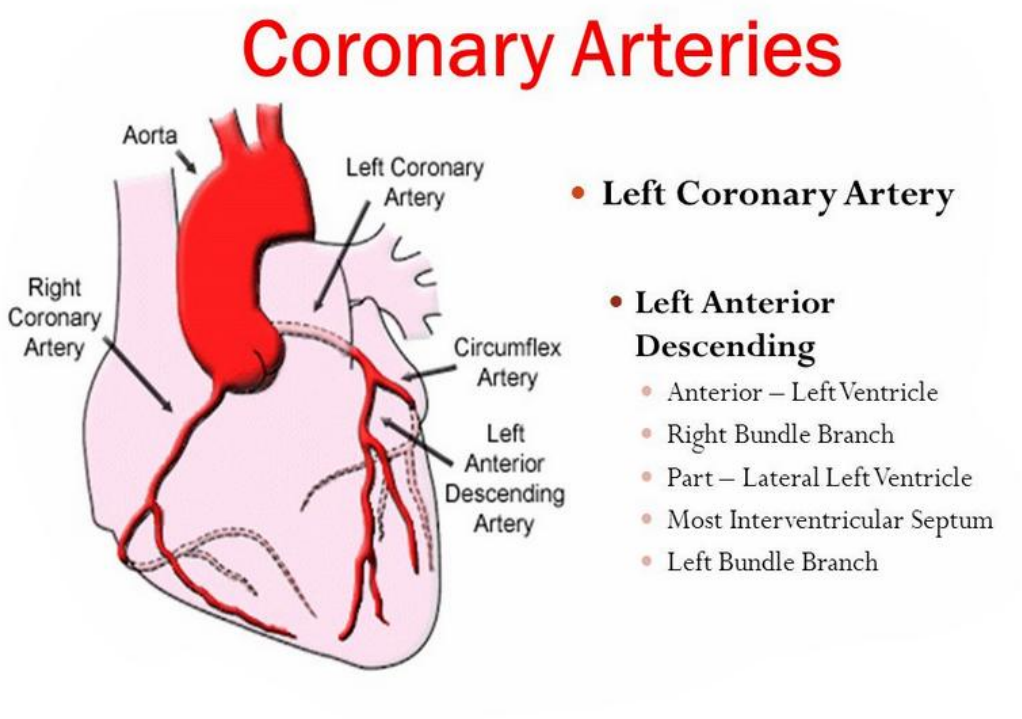
Heart muscle supplied by 2 coronary vessels namely rigt and left coronary vessel, which are the first branches of aorta. Arteries surround the heart in the manner of crown (hence the name latin word corona means=crown).



Picture: 3 Blood supply of the Heart

Right coronary vessel supplies whole of the RV and posterior part of the LV. Left coronary vessel supplies mainly the anterior and lateral

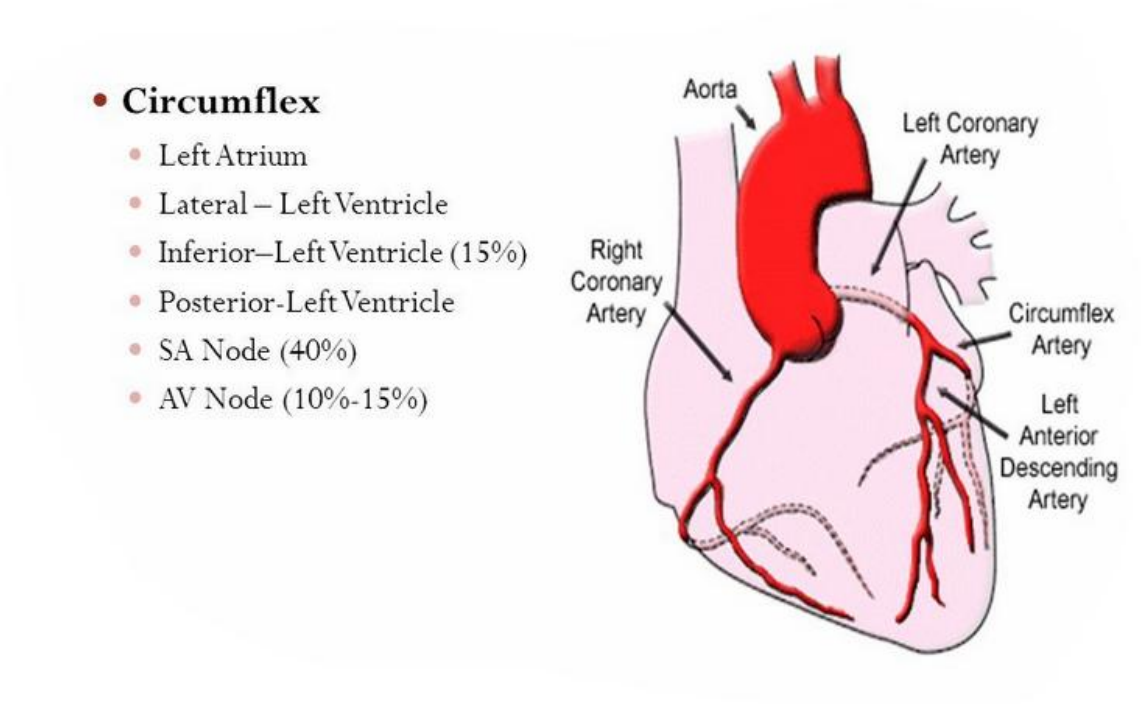
parts of the LV. There are many variations in diameter of coronary vessels.



Picture: 4 LAD and its branches

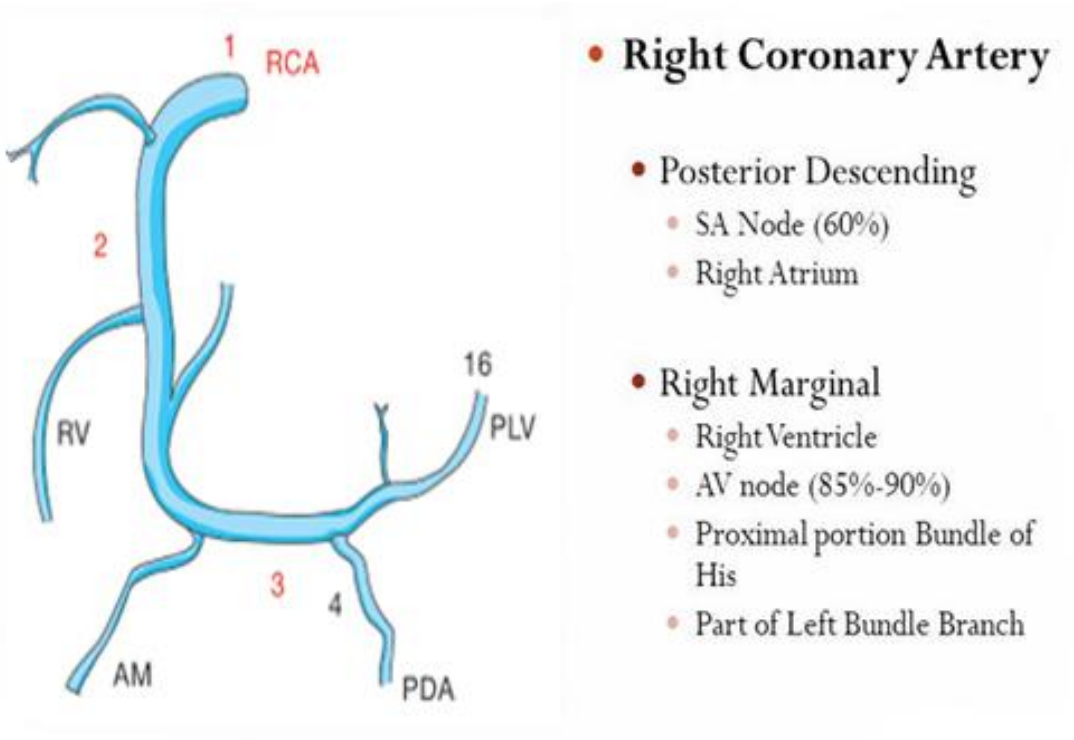
The left coronary vessel originate from left coronary cusp or sinus of the aortic valve. It divides in to LCx branch and left anterior descending branch in the AV sulcus. [10]

The LAD branch runs down in the anterior part of LV and reaches inferior margin and give anastamostic branches to diagonal branch of right coronary vessel. Septum and anterior heart are mainly supplied by this branch.

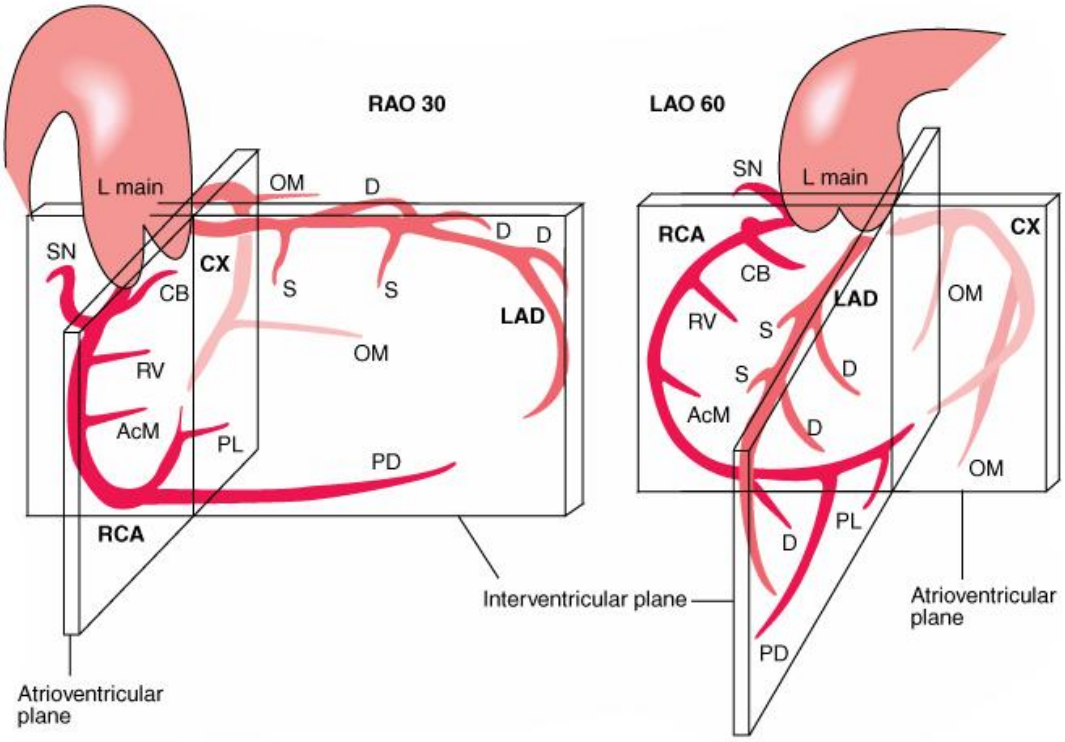


Picture: 5 shows LCX and its branches

The left circumflex vessel runs through the AV sulcus turn around the AV sulcus and give branches for anastamosis with right coronary vessel. Most of the lateral wall and part of anterior wall are supplied by this vessel. [10]



Picture:6 RCA and its branches



Picture: 7 shows RCA, LAD, LCx with all branches

Anatomical variations:

1. In fifty to sixty percent of persons, the RCA is larger and its called right dominant and supplies more parts of the heart than left vessel.

2. In fifteen to twenty percent of persons, left vessel is larger and its called left dominant.

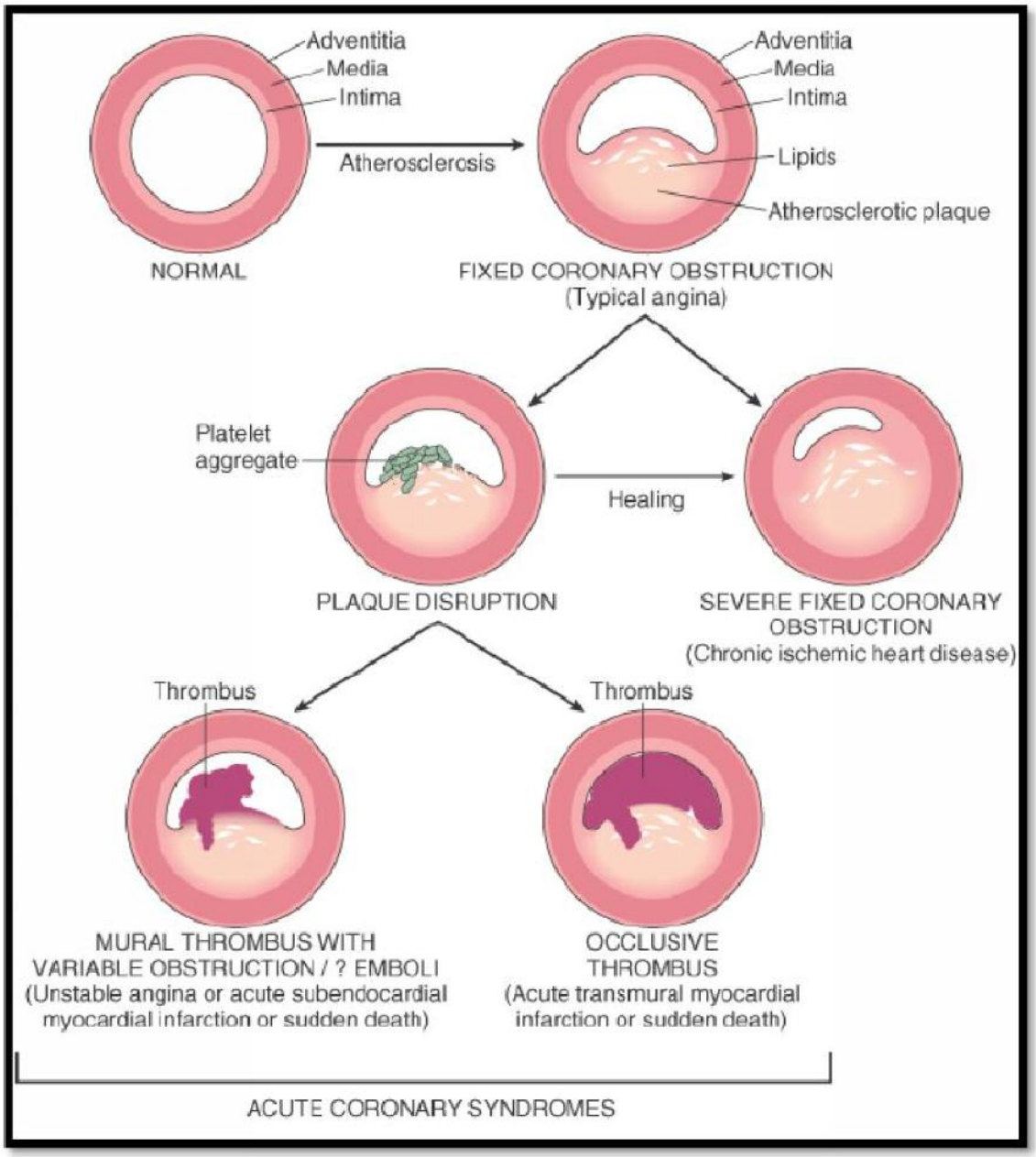
3. In twenty to thirty percent of persons, both arteries supply almost equal[11]

PATHOPHYSIOLOGY:

The blood supply to the myocardium is decreased due to blockage of coronary vessels leads to hypoxia there is O2 supply is diminished to hear muscle. Cardiac ischemia due to prolonged diminution of O2 leads to incomplete removal of metabolites. There is an imbalance between O2 supply and O2 demand leads to ischemia.

Stable atherosclerotic plaque leads to ischemia and its produce pain in the chest. Any dislodgement from this plaque or clot due to spasm of coronary vessels leads to ACS. The blood flow is very much decreasedin this condition. Chance of MI due to non atheromatic cause is very less.

The first step and initiating the atheromatous lesion formation is endothelial injury. Lesion initiation occurs when endothelial cells, activated by RF such as hyperlipoproteinemia, it become more permeable and express the chemottractant molecules that recruits leucocytes, lymphocytes and monocytes. There is release of enzymes, cytokines and growth factors will leads to focal necrosis of vessel wall. The simultaneous repairing process will occur after necrosis and it is called fibrosis. The ECF lipids are accumulate in the inner layer of the vessel wall. [12]



Picture: 7 pathogenesis of CAD

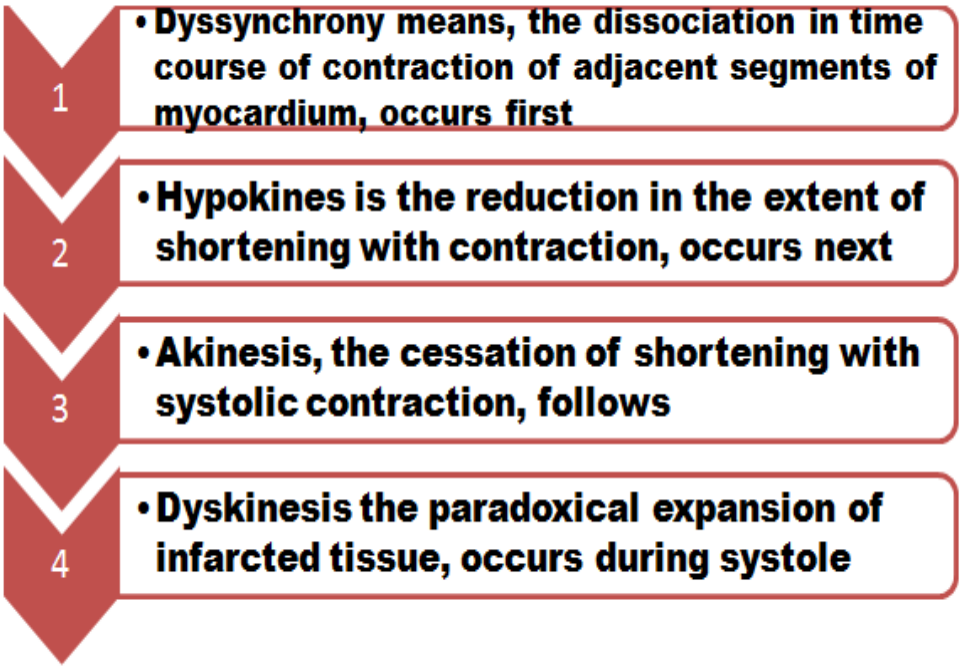
Activation of thrombocytes leads to attached to the sub endothelial adhesion molecules. It has thrombogenic potential and bind with laminin, fibronectin, collagen and binding to GP Ib receptor to form of vWF. Endothelial collagen is a strong stimulator of activation of platelets.

The atheroma plaque contains lipid laden macropages in tunica adventitia release TF (tissue factor) and covert to prothrombin to thrombin. These are also a potent stimulant for activation of platelet. ADP, $TXnA_2$, and serotonin are auto stimulatory agonists of PA. Activated thrombocytes and GP IIb/IIIa both are cross-linked between each other by vWf or fibrinogen in the end common pathway of PA.

The extent of O2 deprivation leads to patient gets the clinical symptoms of ASTEMI. It is depends on plaque stability fixed, fissured and eroded plaque. MI can be manifested as by chest discomfort, breathlessness, specific or nonspecific ecg changes, decreased myocardial contractility, decreasedcardiac output and peripheral blood flow. In case of stable angina, ischemic pain occurs only when O_2 demands beyond the O2 supply seen in incomplete coronary blockage. It is slowly changes into over a time. [12]

When it is rupture and form thrombus which contain platelets. The blood flow to the coronary vessel is decreased and it cause myocardial ischemia or infarction. The demand of O2 and supply of O2 to myocardial tissue, this imbalance determines reversible MI without necrosis (unstable angina) or myocardial ischemia with necrosis.

Acute ischemic condition may leads to impaired contraction of myocardium due to reduced cardiac output. In acute MI, the basic alteration is loss of function of the myocardial tissue. When O2 supply is further reduced to the tissues leads to defective contraction.



When size of the infarct is increase may leads to affect large portion of myocardial segment that case failure of pumping capacity of heart. This leads to LV EDV and LV ESV increase and decrease CO, stroke volume, and BP. When LV pressure is raised, that leads to elevation of left atrial pressure. Chronic elevation leads to pressure changes in the lung capillaries leads to pulmonary edema and CCF. When CCF occurs it leads to reduced CO and decrease blood flow to the brain and kidney.

ST-ELEVATION MI

The main symptom of ischemic heart disease is pain in the chest, and the history should ask about character of the pain such as radiating to shoulder, arm, upper back, jaw and chin and ask about duration of the pain and quality of the pain. Patients presented with any history of breathlessness, sweating, vomiting and palpitations are noted.

Picture: 8 closed fist sign is called LEVINE sign

Clinical symptoms of acute MI frequently will be elaborated as discomfort of the chest rather than pain. Classic symptoms of angina are tightness in the chest, squeezing, feel heaviness in the chest and

compressing in nature. Less common symptoms are sharp stabbing or knife like pain. The classic location is pain in the central area of the chest, left side chest and radiating to neck, arm and back. [13]

Any strain or stress or cold climate can cause angina pain. Ischemic pain or anginal pain is lasting less than ten minutes, and sometimes lasting ten to twenty minutes. Stable one is relived by two to five minutes of rest and NTG. In AMI is mostly associated with intolerable pain in the chest associated with discomfort in the chest. This also associated with sweating, nausea, breathlessness and easy fatigue.

30% of the ASTEMI subjects have atypical symptoms clinically not associated with specific symptoms of MI. Some patients gets silent MI. Patients with atypical symptoms has worse prognosis than typical one. Female patients and aged patients are usually to have atypical pain or nonspecific pain. and the elderly are more likely to have atypical presentations.

RISK FACTORS:

NON MODIFIABLE RF:

- Age
- Sex-male
- Positive family history
- Detection polymorphism in the ACE gene

MODIFIABLE RF

- High levels of serum lipids
- History of Smoking
- DM
- SHT
- Chronic ethanol use
- Sedentary life style
- high fibrinogen, factor 7
- c-RP
- high homocystein level in the blood, gout
- type A personality
- obese individuals
- soft water,OCP

DIAGNOSIS OF ASTEMI:

PHYSICAL EXAMINATION:

The physical examination is quite nonspecific in ACS diagnosis and it is not useful in differentiating patients with ACS with non cardiac cause. Subjects with ASTEMI may appear deceptively well without any clinical signs of distress or may be uncomfortable, pale, cyanotic, and in respiratory distress. Most important thing in physical examination is to identify the consequences of acute ischemia. The patient may have normal or bradycardicrythm, tachycardiacrythm, or irregular pulses. Bradycardia are more common in patients with IWMI. If heart block and bradicardiac rhythm are associated with AWMI will be bad prognostic sign. Sometimes BP may be low (hypotension) or high (hypertension). In AMI, there is impaired contraction of the myocardium leads to S1 and S2 are decreased. In fifteen to fifteen percentage of subjects with STEMI have S3 gallop. In subjects with longstanding SHT or dysfunction of the myocardium have 4^{th} heart sound (S4). S3 also present in patients with MI with cardiac failure. Sometimes patient with ASTEMI will have systolic murmur due to impaired function of the papillary muscle due to infarction. It leads to acute MR with cardiac failure and also rupture of LV leads to VSD. Differential diagnosis of ASTEMI like dissection of the aorta can be differentiated by examining the unequal pulse and early diastolic murmur due to AR. [12]

ASTEMI patients developed cardiac failure having signs of HF like bilateral basal crepitations of the lungs, sometimes presented with s3 gallop along with LV failure. They also have signs of CCF, elevated JVP, swelling of legs, distension of neck vein and hepato jugular reflex.

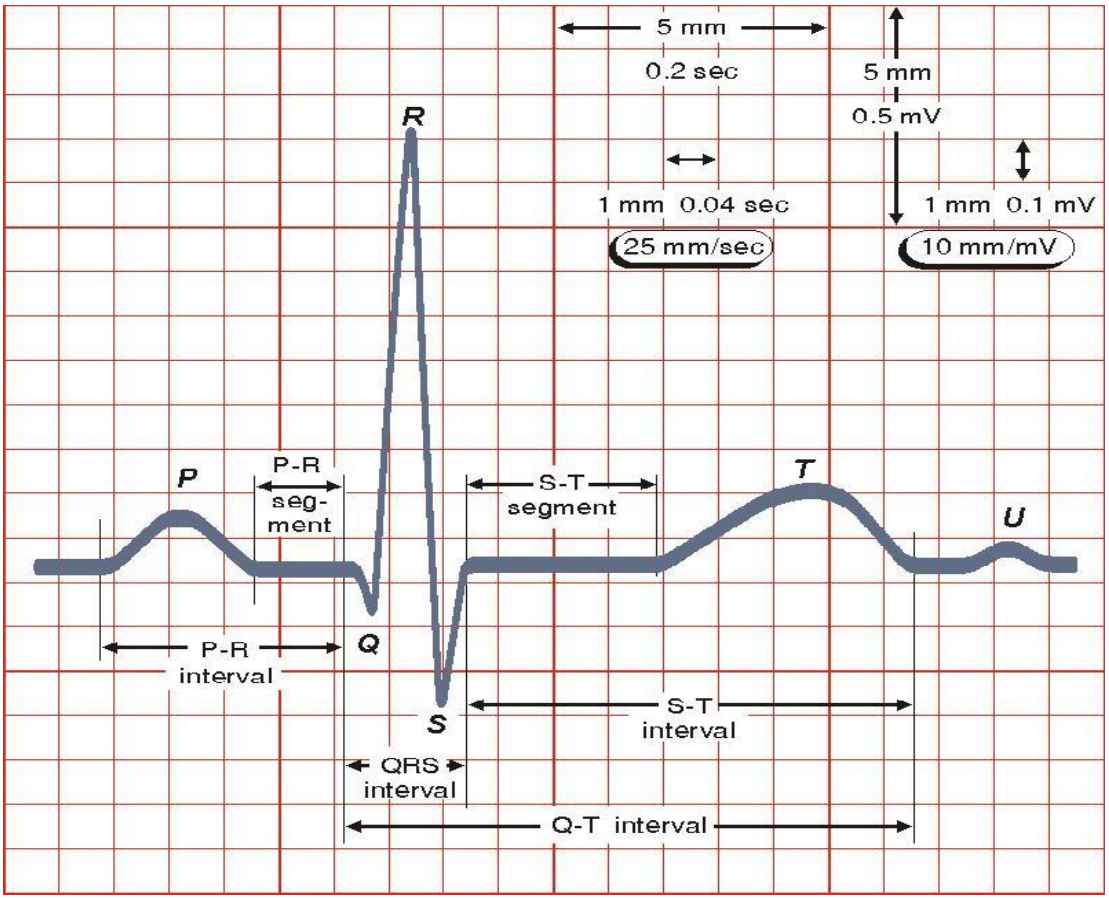
ELECTROCARDIOGRAM:

The electrocardiographic method is the most useful method for diagnosing ASTEMI subjects. It is a non invasive less expensive method. The twelve leads electrocardiogram can gives information to diagnose the problems and prognosis.

The myocardial tissue necrosis can cause depolarization changes in the QRS complex. It is differentiate the MI with ASTEMI from Non ST segment elevation MI. ST segment elevation more than one millimeter in 2 or more leads is classified as ASTEMI, an indication for thrombolytics treatment. Non STEMI ACS may present with ST segment depression or T wave inversion or transient ST segment elevation. The degree of ST segment depression had a prognostic value. Patient with more than0.2mv had increased mortality.

ECG will inform us about other parameters like HR, duration of the QRS complex, conduction abnormalities, and presence or absence of a old infarction which play an important role in the treatment and prognosis of the STEMI patients. Following thrombolytic treatment the

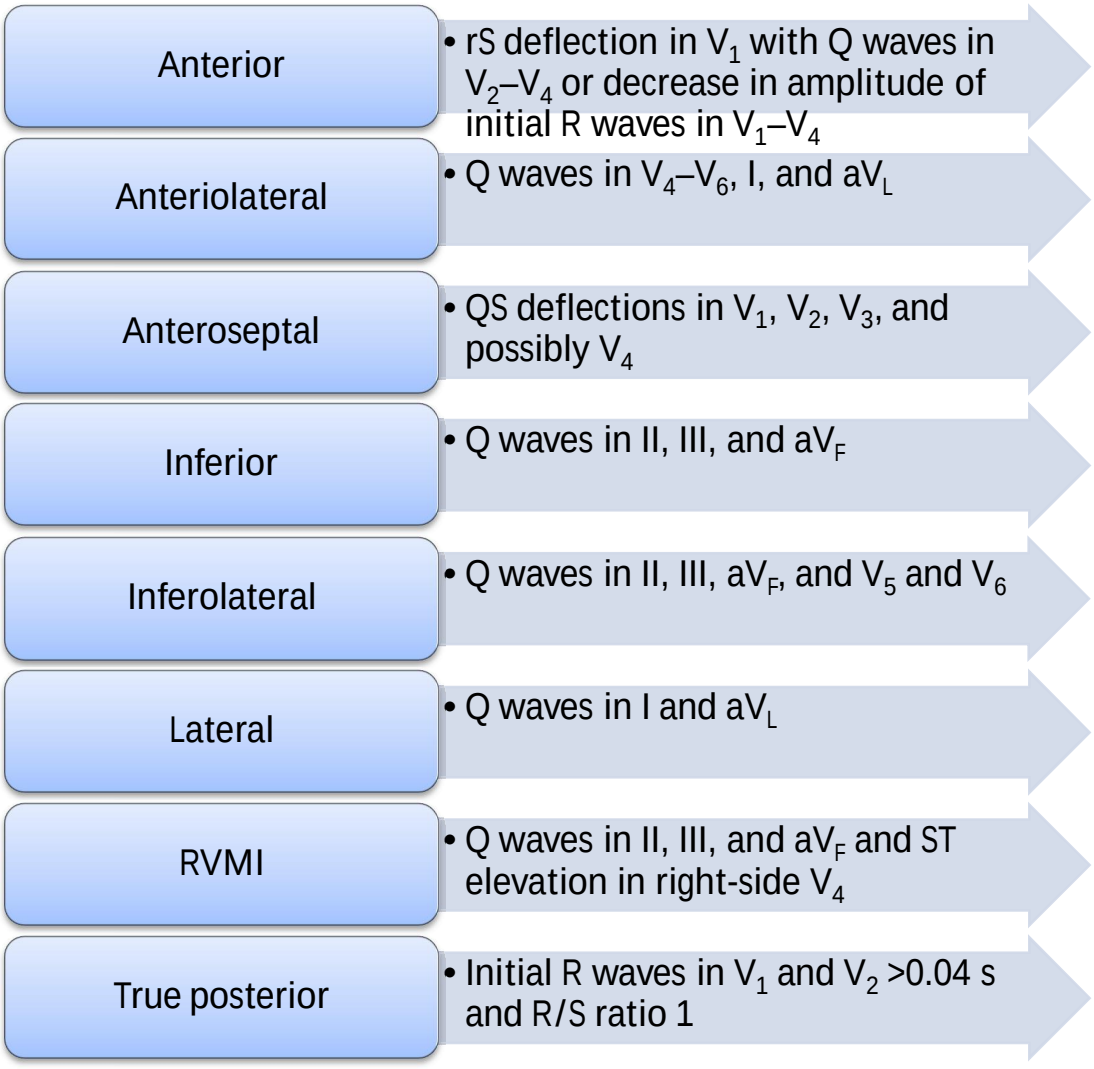
electrocardiogram can inform us about the failure of thrombolytic treatment help us to select which STEMI patient should receive a rescue percutaneous intervention.



Picture: 9 Normal ECG

SPECIFIC ELECTROCARDIOGRAM:

The electrocardiogram gives us, information about the site of blockage of coronary vessels in patient with ASTEMI. Electrocardiographic leads are useful in localization of ischemia or site of infarction. [12]

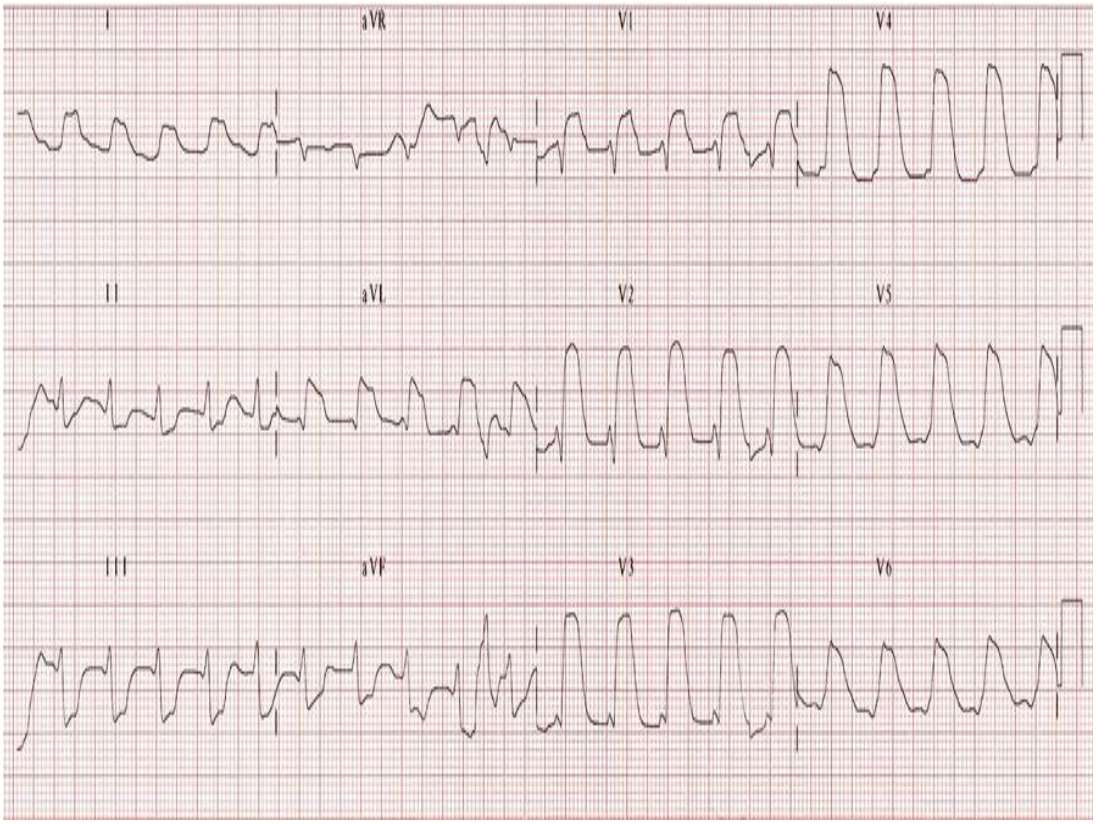


Picture: 10 Types of ASTEMI

ANTERIOR WALL MI:

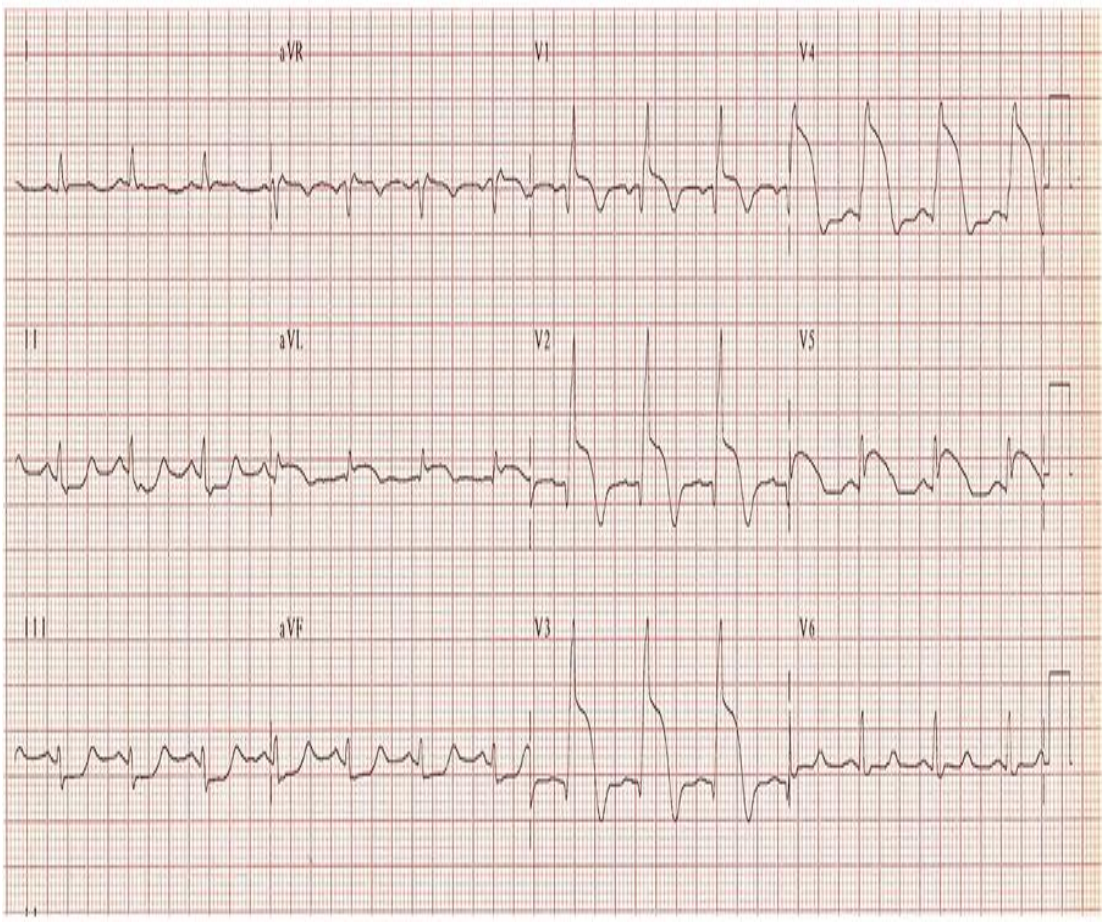
ST segment elevation is present in chest leads and depression lead III and avF has reciprocal ST segment depression. If this changes absent in lead avF indicates blockage of distal LAD vessel.

Anterior-ST segment elevation in chest leads from v2 to v5 is called as anterior wall MI, if there is ST segment elevation in chest leads from v1 to v4 is called as ASMI.



Picture: 11 AWMI –tomb stone pattern

Anterolateral MI means ST segment elevation in chest leads from v3 to v6 and lead I and avL, v5 and v6. ST segment elevation especially in leads I and aVL and fewer changes in chest leads occur in the blockage of D1 branch of LAD, while ST segment elevation mainly in chest leads v1 and v2 with reciprocal depression in limb leads II,III,aVF,V5 and V6 occurs when main S1 of LAD is occluded.

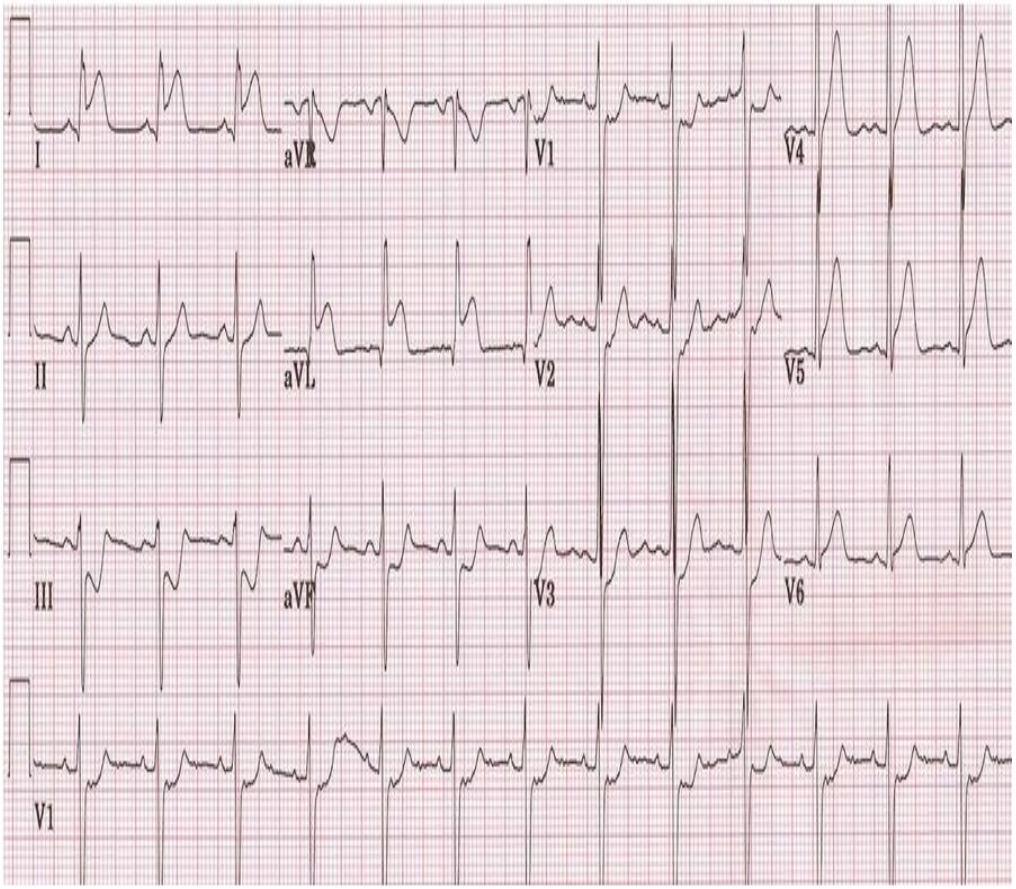


Picture: 12 ASMI

LATERAL MI OR APICAL MI:

ST segment elevation occurs in chest leads v5 and v6 with reciprocal depression in limb lead III and aVF and occasionally in chest lead v1.

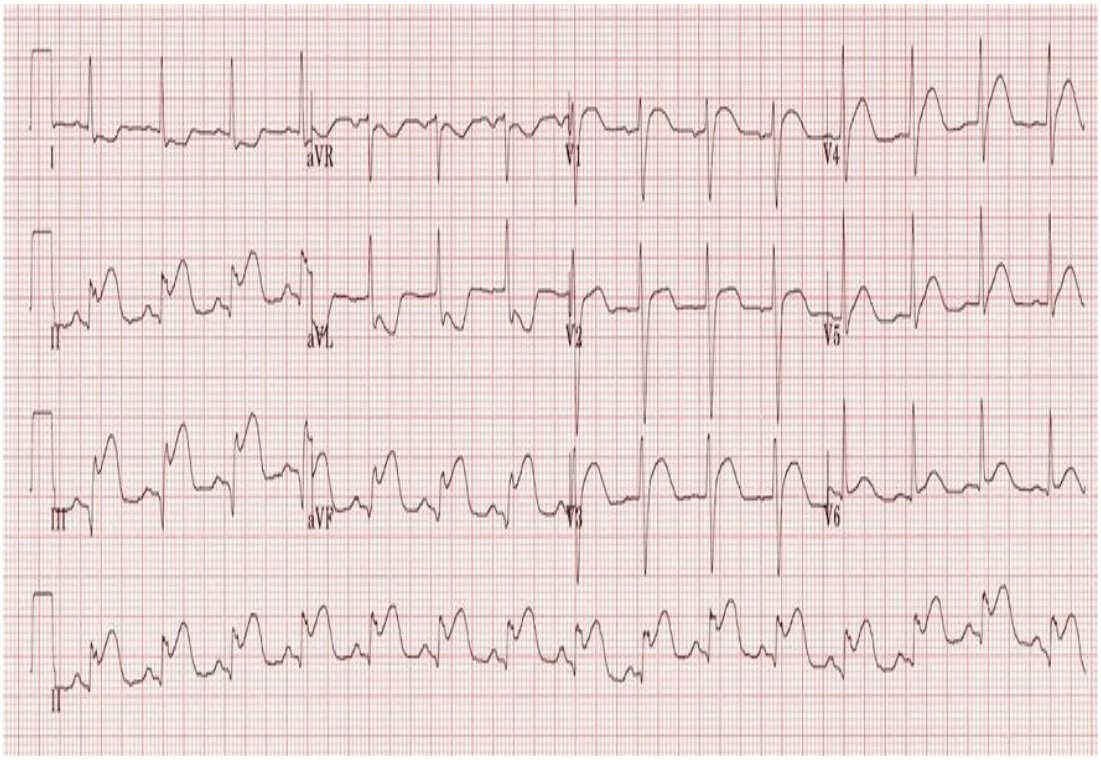
High lateral MI means ST elevation in lead I and aVL. Patient with high lateral MI is less common than other STEMI.



Picture: 13 High lateral ASTEMI

INFERIOR MI/RVMI:

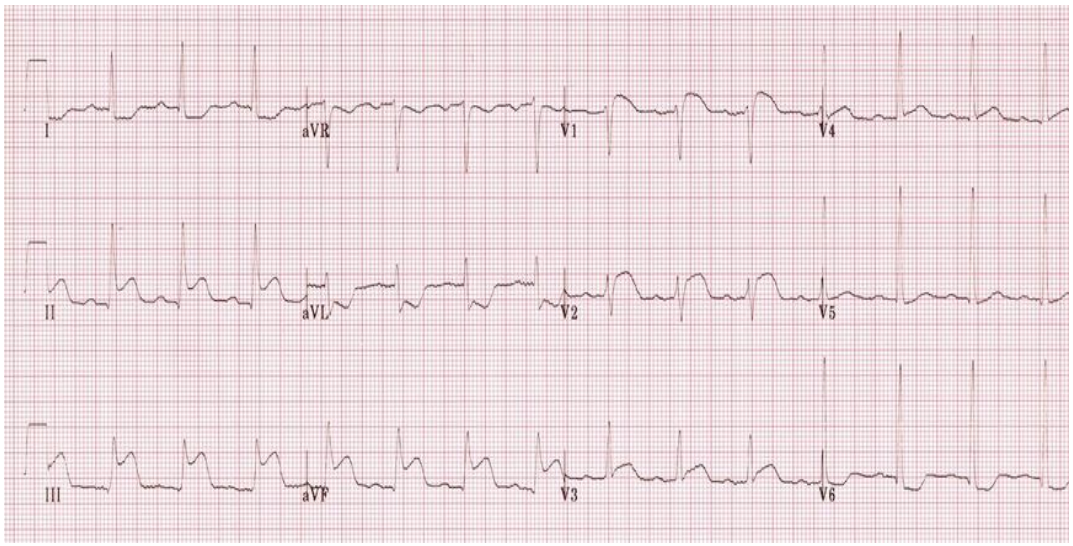
ST segment elevation occurs in limb leads II,III, and aVF with reciprocal ST segment depression present in limb leads I and aVL and one or more chest leads like v2-v3. This changes are absent in chest leads with ST segment elevation in v1, we should suspect RVMI. Presence of additional ST segment elevation in lateral chest leads v5, v6 or in leads I and aVL represents the blockage of LCX and it is called Inferolateral MI.

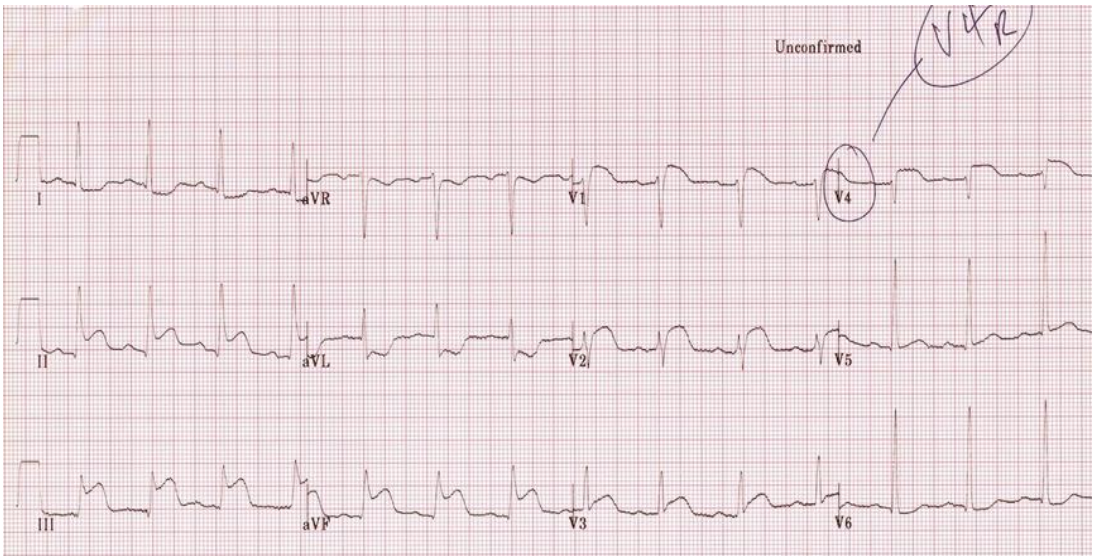


Picture: 14 IWMI

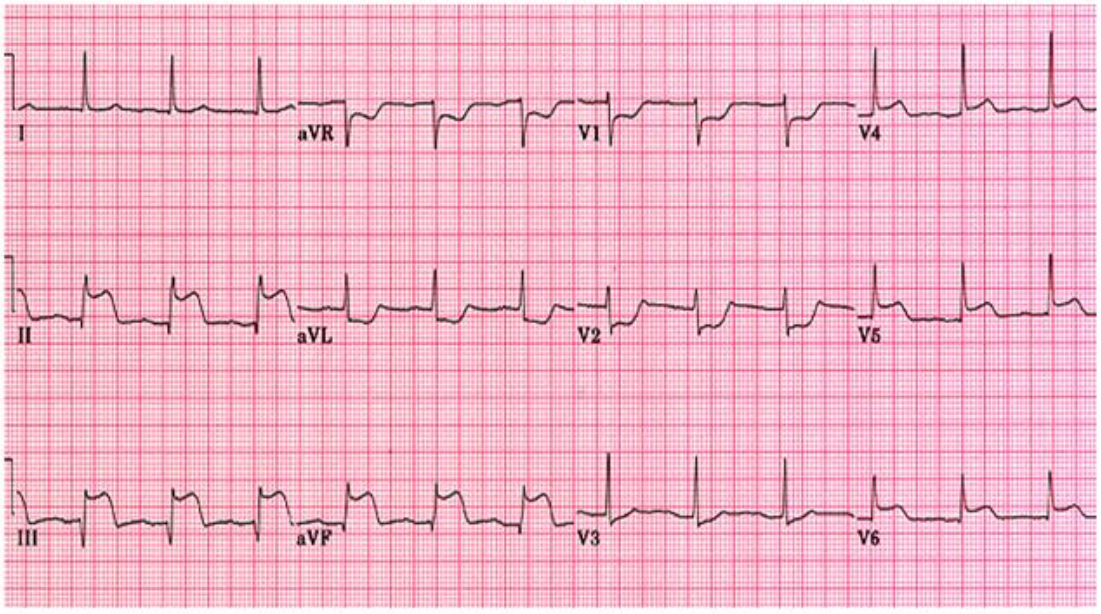
RVMI:

ST elevation in leads II, III and aVF and ST segment depression in reciprocal leads I, aVL and absence of ST depression in V2, v3. ST elevation present in right sided lead V4R.





Picture: 15 shows RVMI

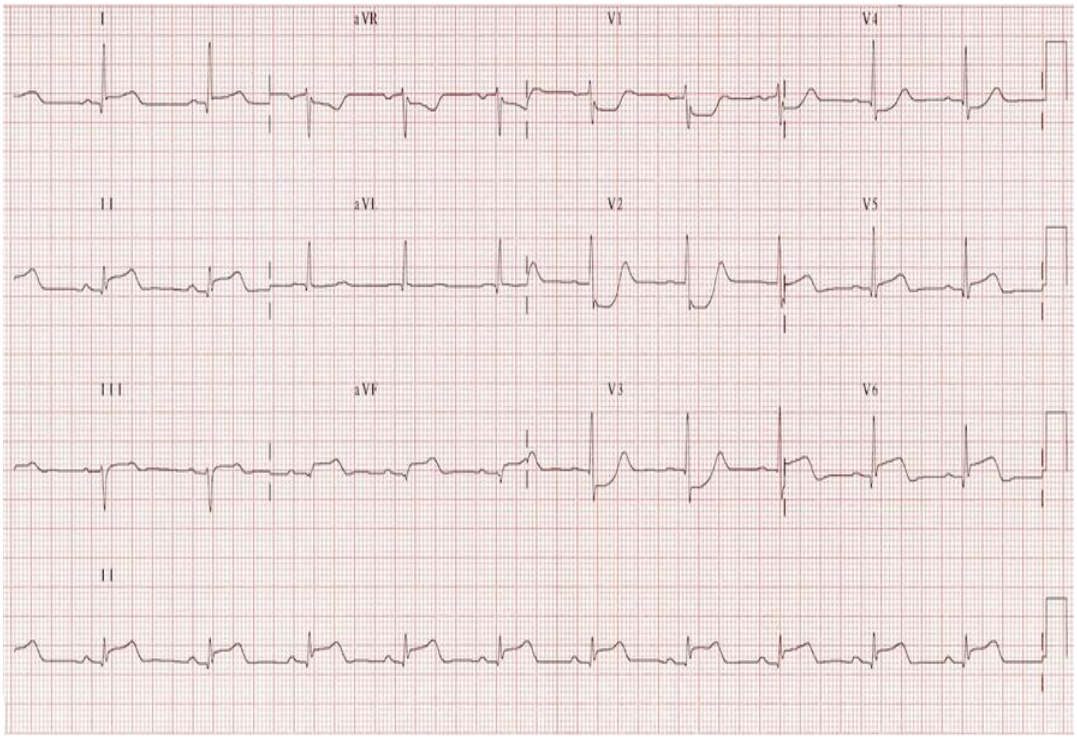


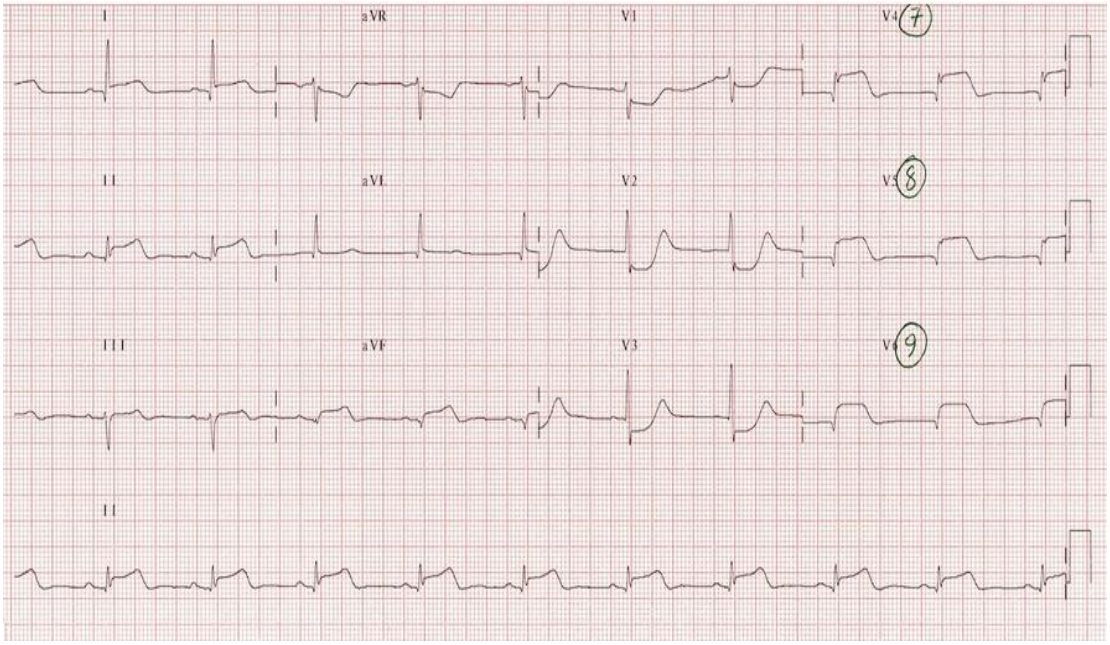
Picture: 16Inferolateral ASTEMI

POSTERIOR MI:

Increase R wave amplitude in V1 and V2, ST segment depression with upright T waves

Q waves and ST segment elevation in posterior chest leads v7-v9, Posterior MI is always combined with IWMI.





Picture: 17 shows posterior ASTEMI

LVMI WITH PAPILLARY MUSCLES (PM) INVOLVEMENT:

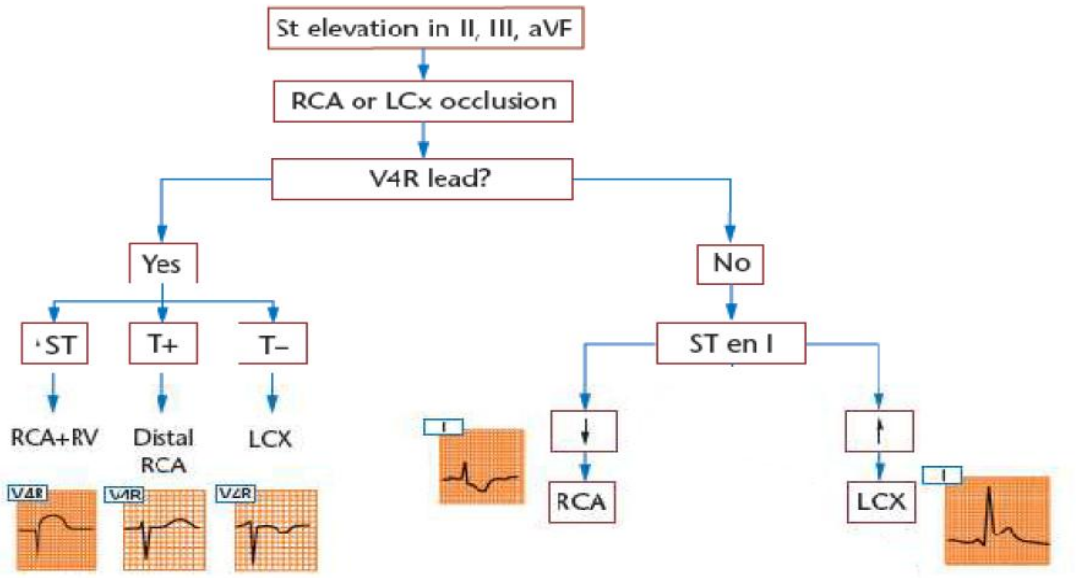
Along with ST segment-T changes in LV leads, ST segment depression in inferior leads are indicates anterolateral PM infarction, while ST segment depression in lead I or aVL is seen in posteromedial PM infarction.

DIAGNOSIS OF CULPRIT VESSEL:

The electrocardiogram gives information about the site of blockage of coronary vesseles in subjects with MI ischemia. Oc Right coronary vessel blockage causes inferior STEMI, posterior STEMI, inferoposterior STEMI, inferolateral STEMI, and RVMI. Blockage of Lcx vessel causes posterior STEMI and lateral STEMI. Blockage of dominant LCx artery is cause inferoposterior STEMI or posterolateral STEMI. [12]

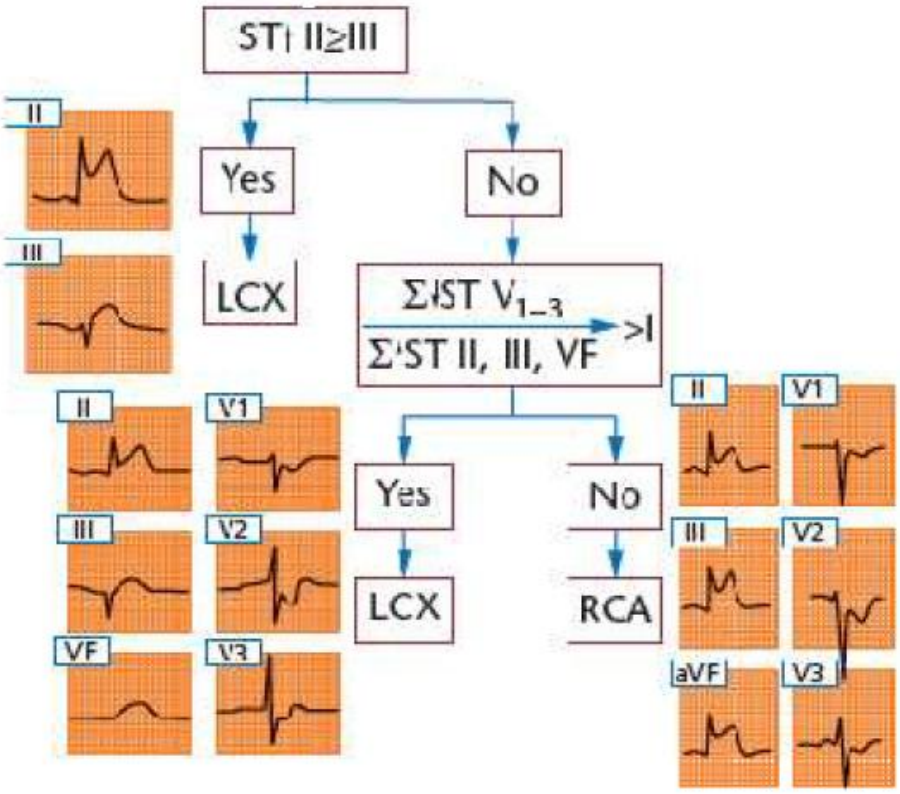
Blockage of LAD branch causes ASMI and AW MI. Involvement proximal LAD branch can cause bundle branch block and LAFB.

PREDICTING RCA OR LCX BLOCKAGE IN IW:



Picture: 18(a) culprit artery diagnosis in IWMI

ST segment elevation in lead III is more than lead II is represents RCA blockage and ST segment elevation in lead II is more than III represents LCX blockage. In inferior infarction, ST segment elevation in lead III is more than II, and combined with ST segment elevation in v1 is a powerful predictor of proximal right coronary vessel blockage.

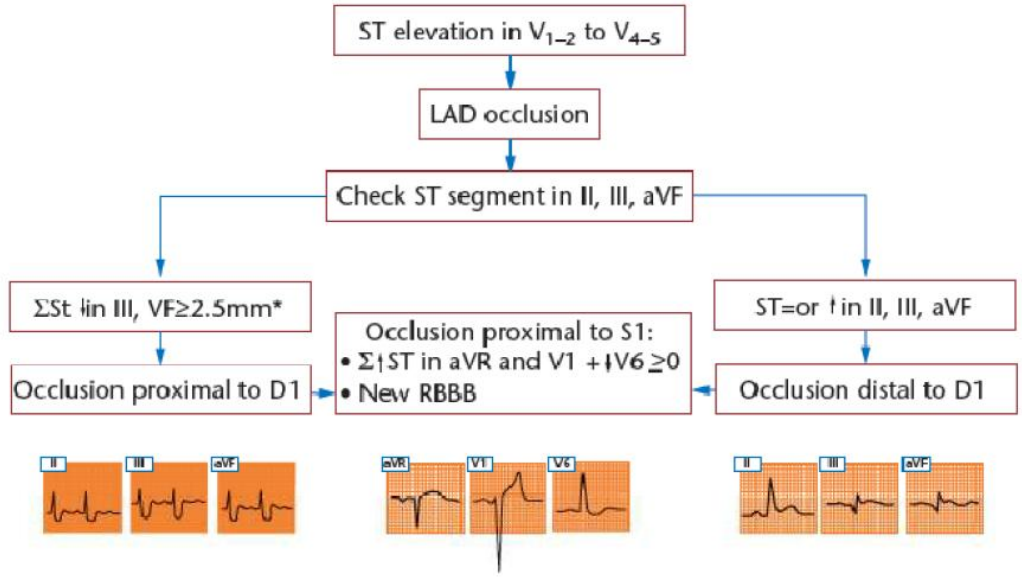


Picture: 18(b) culprit artery diagnosis in IWMI

Inferior MI with ST segment elevation in lateral chest leads v5 and v6 or in limb leads I and aVL represents LCX blockage. Inferior MI with ST segment depression in V1-V3 is more frequently associated with LCx vessel than right coronary vessel blockage. Inferior MI with reciprocal changes like ST segment depression in aVL predict LCX blockage, while reciprocal changes in chest leads fromV4 toV6 predict the multi vessels disease.

PREDICTING LAD BLOCKAGE IN ANTERIOR MI:

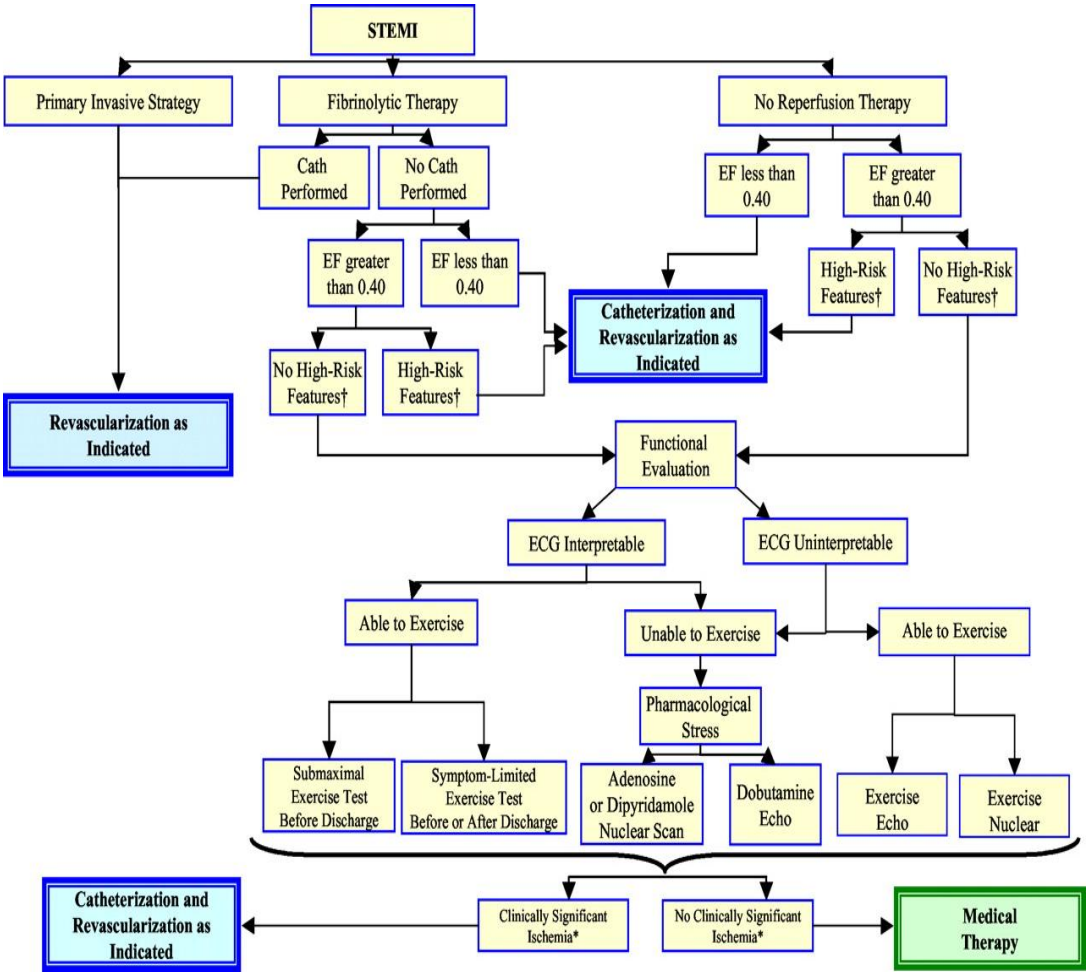
ST segment elevation in chest leads combined with ST segment depression more than or equal to 1mm predicts LAD proximal to branch S1 (serptal) or diagonal branch D1. ST segment elevation in V1 and aVR associated with complete RBBB and ST segment depression in V5 predicts blockage of LAD proximal to S1. Abnormal Q waves in aVL and ST segment elevation in I and aVL predicts blockage of proximal to D1.ST segment elevation in chest leads with presence of abnormal Q waves in from V4 to V6 represents LAD blockage distal to S1 or D1. ST segment depression in aVL combined with precardial ST segment elevation represents LAD blockage distal to D1 branch.



Picture: 19 culprit artery diagnosis in AWMI

ASTEMI MANAGEMENT:

Categorize the patients with STEMI for giving appropriate treatment depends upon the infrastructure and facilities of the treatment centre.



Picture: 20 flow chart shows STEMI management algorithm

KILLIPS CLASS:

CLASS	CLINICAL DESCRIPTION	MORTALITY RATE
1.Normal	No clinical evidence of LV failure	3-5%
2.slightly reduced	Mild to moderate LV failure	6-105
3.Abnormal	Severe LV failure,pulmonary oedema	20-30%
4. severely Abnormal	Cardiogenic shock, tachycardia, hypoxia.	>80%

Table shows STEMI presented with symptoms are classified

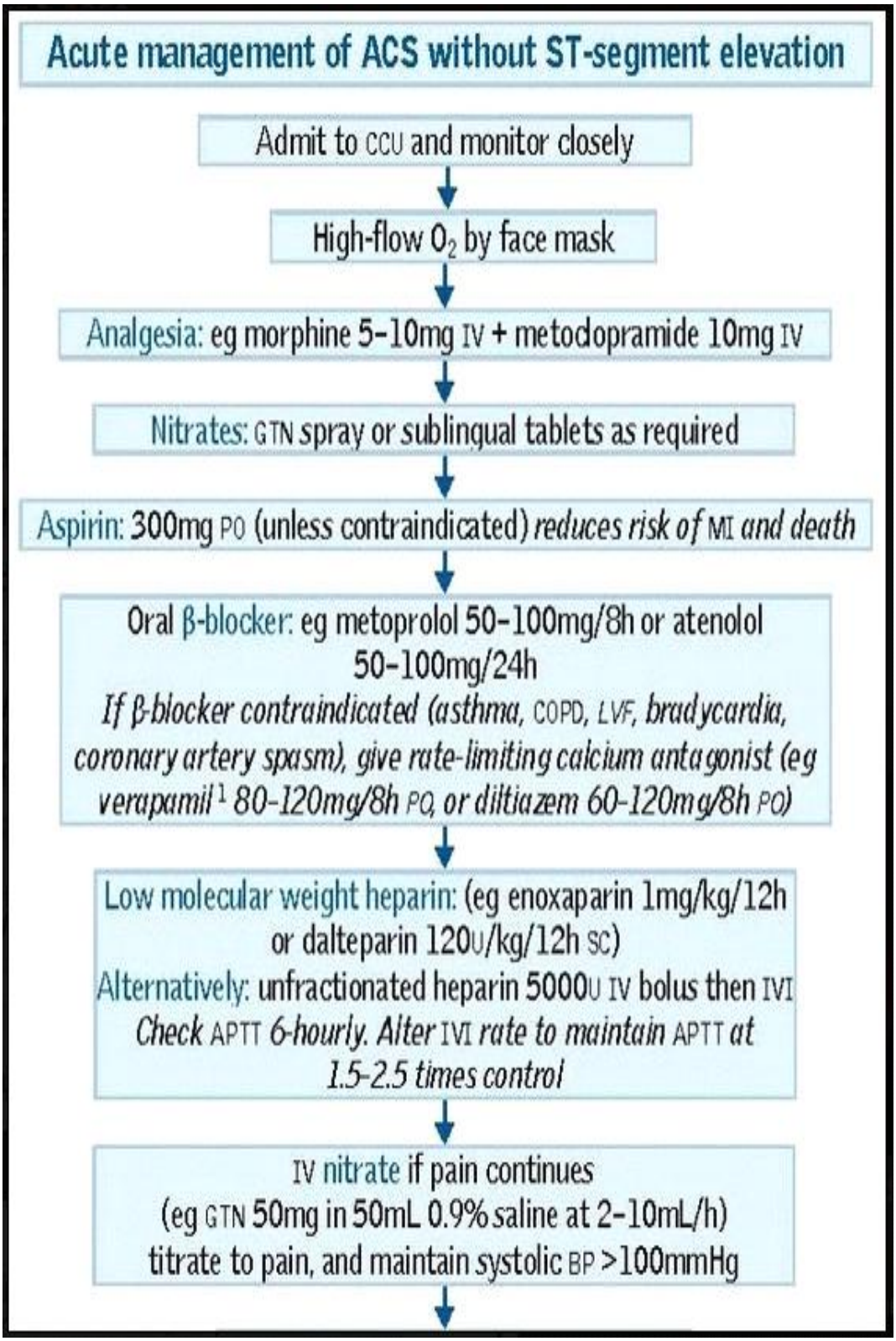
TIMI Risk Score for STEMI

Historical	
Age 65-74	2 points
≥ 75	3 points
DM/HTN or angina	1 point
Exam	
SBP < 100	3 points
HR >100	2 points
Killip II-IV	2 points
Weight < 67 kg	1 point
Presentation	
Anterior STE or LBBB	1 point
Time to rx > 4 hrs	1 point
Risk Score = Total	**(0 -14)**

Risk Score	Odds of death by 30D*	
0	0.1	(0.1-0.2)
1	0.3	(0.2-0.3)
2	0.4	(0.3-0.5)
3	0.7	(0.6-0.9)
4	1.2	(1.0-1.5)
5	2.2	(1.9-2.6)
6	3.0	(2.5-3.6)
7	4.8	(3.8-6.1)
8	5.8	(4.2-7.8)
>8	8.8	(6.3-12)

*referenced to average mortality
(95% confidence intervals)

Timi score used to assess the mortality in STEMI patientsto assess the risk of mortality . [12]


Acute management of ACS without ST-segment elevation
Admit to CCU and monitor closely
High-flow O_2 by face mask
Analgesia: eg morphine 5–10mg IV + metoclopramide 10mg IV
Nitrates: GTN spray or sublingual tablets as required
Aspirin: 300mg PO (unless contraindicated) reduces risk of MI and death
Oral β-blocker: eg metoprolol 50–100mg/8h or atenolol 50–100mg/24h
If β-blocker contraindicated (asthma, COPD, LVF, bradycardia, coronary artery spasm), give rate-limiting calcium antagonist (eg verapamil[1] 80–120mg/8h PO, or diltiazem 60–120mg/8h PO)
Low molecular weight heparin: (eg enoxaparin 1mg/kg/12h or dalteparin 120U/kg/12h SC)
Alternatively: unfractionated heparin 5000U IV bolus then IVI
Check APTT 6-hourly. Alter IVI rate to maintain APTT at 1.5–2.5 times control
IV nitrate if pain continues
(eg GTN 50mg in 50mL 0.9% saline at 2–10mL/h)
titrate to pain, and maintain systolic BP >100mmHg

ANTIPLATELETS: ASPIRIN

162 to 325 mg of aspirin, it inhibits the platelet aggregation and block the production of TxA2 and it causes prevention of vasoconstriction. It is contraindicated in active APD, bleeding diathesis and patient allergy to aspirin.

O2 THERAPHY:

O2 should be given at 2 to 4 L/min by nasal mask to maintain SaO2 level more than 90%. We noted the signs of hypoxia such as confusion, altered sensorium, restlessness pallor, and cold skin. Should treated with high flow oxygen can reduce the ischemic pain symptoms.

NTG:

NTG causes vasodilatation mainly veins than arteries and it reduces the both preload and after load of the heart. Nitroglycerin is not given in patients SBP is less than 90 mmhg, heart rate (HR) less than 50 or more than 100 in the absence of HF. NTG should not be given along with phosphodiesterase inhibitor within 1-2days it aggravates hypotension. It can be given by oral, nasal, sublingual and IV infusion. [12]

MORPHINE:

Morphine sulfate is the opioid of choice for chest pain especially in ASTEMI patients who are not relieved by NTG. It may be given at starting dose of a 2-to-4-mg IV and it can be given repeat dose in every fifteen minutes until the patient feels better. It can cause both arterial and venodilator and decreases the preload and afterload in addition to analgesic properties in ASTEMI patients. It can cause depress the respiratory system and also reduction of BP. We monitored the BP, RR, and O2 saturation carefully.

COPIDOGREL:

Clopidogrel can be given an initial dose of 300mg or 600 mg then followed by 75mg every day. It is also given before PCI or stent is placed and continue this drug as 75mg OD daily for at least 12 months. It inhibits platelets clumping and can be given to ASTEMI patients with a patient known allergic to aspirin. It should not be given if bypass surgery is planned within the next 5 to 7 days. It increases a risk of more bleeding during operation.

STATINS:

It has pleomorphic activities. It should be given at loading dose atorvostatin 80mg followed by 40 mg once daily at night time. It lowers the cholesterol by inhibition of HMG-CoA reductase. It also reduces the inflammation and protect the cardiac myocytes.

BETA BLOCKERS:

It reduces the myocardial O2 demand and decrease the ventricular strain. It also reduces mortality and prevent complications due ASTEMI. It decreases the workload of the heart in patients with dynamic obstruction of the LVOT. Initial dose should be given as 2 to 5 mg IV in every five minutes followed by 25 to 50 mg oral BD per day. [12]

Contraindications:

Systolic blood pressure less than 90 mm Hg

hypotension

Cardiogenic shock

Bradycardiacrythms

Heart block , II and III degree block

Asthma

PAD

CHF

GP IIB/IIIA INIBITORS:

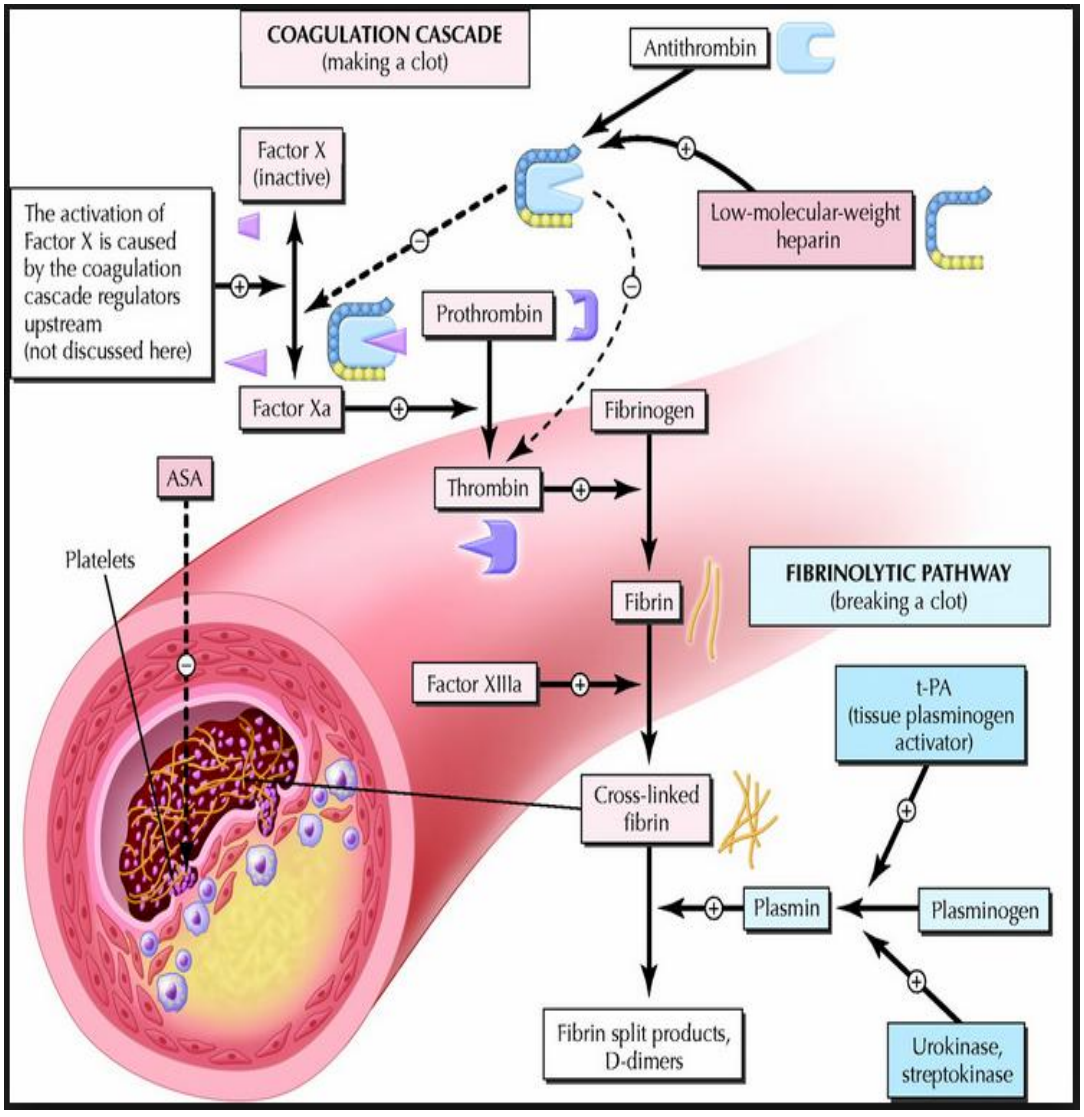
Many different GP IIb/IIIa antagonists are available. Abciximab is a chimeric antibody it binds irreversibly to the GP IIb/IIIa antagonists Eftifibatide is a synthetic heptapeptide that binds reversibly to the GP IIb/IIIa receptor. Tirofiban is a synthetic small molecule reversible binding to the GP IIb/IIIa. It should be given as an IV infusion .Reversal of platelet inhibition after cessation of infusion is more rapid with the polypeptide or small molecules.

SPIRANOLACTONE:

Arrange for an ECHO to be done within 24 hrs of admission. Patients with HF and the LV EF is less than or equal to 40%, consider an aldosterone antagonist such as spiranolactone 25mg od should be given.[12]

HEPARIN:

Heparin is a specific antithrombotic agent.Thrombin generation plays a main role in pathogenesis of coronary vessel thrombosis. Thrombin converts soluble fibrinogen to insoluble fibrin and activates coagulation factors V and VIII. It activates factor XIII which stabilizes colt formation by augmenting fibrin cross linking. It also plays as a platelet antagonist.



Picture: 21 - Drugs interfere with clot formation

UFH:

Unfractionated heparin has molecular weights varying between 2000 and 20,000.The different size molecules have different effect on the clotting system. Heparin complexes with antithrombin III and this complex inactivate factor thrombin and activated factor X. The heparin antithrombin III complex is not effective against clot-bound thrombin. It requires careful monitoring and dose adjustment .

The weight based regimen is given to patients with ASTEMI. The initial loading dose is 60 to 70Units per kilogram and maximum dose is 5000units, followed by an infusion of 12 to15units per kilogram per hour and maximum dose is 1000units per hour for 24 to 48hrs and activated thromboplastin time should be maintained at 60-70seconds.

LMWH:

The low molecular weight heparin has greater bioavailability and lower protein binding. It has a longer half life .It achieve a more reliable anticoagulant effect. They can be given in a fixed dose subcutaneously once or twice a day. In patients whom CABG is planned, it should be with held and shifted to UFH in the 12 to 24 hours before surgery. If patients less than 75years, initial dose should be given as 30 mg IV bolus followed by 1 mg per kg subcutaneous once a day not more than 100mg. Maintenance dose is 1 mg per kg subcutaneous q12hr. If age of the

patients more 75 years, initial dose should not be given, maintenance dose is 0.75mg per kg subcutaneous every 12 hours. It should be given along with aspirin. In conjunction with fibrinolytic therapy, enoxaparin should be given before 15 minutes and after 30 minutes of thrombolytic therapy. LMWH may be given duration of 8 days or until discharge. [12]

DIRECT THROMBIN INHIBITORS:

These agents are bind to the catalystic site of thrombin and bind to thrombin in clot and resistant to agents which degrade heparin. A peptide which is derived from medicinal leech called as Hirudin. It consist 65 aminoacid and is one of the most potent natural anticoagulant. Hirudin increased chance of intracranial bleeding compared to heparin.

Bivalirudin may reduce the short-term risk of postischemic complications relative to high-dose UFH in patients PCI for unstable or postinfarction angina, but benefits do appear to persist long term. Similarly, Inogatran does not appear to offer benefits over UFH for the treatment of patients with ACS. Thus, at this time, there is no clearly defined role for direct thrombin inhibitors in the medical management of ACS patients. [12]

REPERFUSION THERAPHY:

Fibrinolytics therapy and PCI are used in reperfusion therapy in patients diagnosed with ASTEMI. The aim of fibrinolytics therapy or PCI is to restore blood flow to the ischemic myocardiam and prevent further complications of acute coronary syndrome .

FIBRINOLYTICS THERAPHY:

Fibrinolytics are dissolving existing thrombi by converting plasminogen to plasmin and degrading fibrin clots. The most commonly used in thrombolysis are streptokinase, alteplase (recombinant tissue–type plasminogen activator [rt-PA]; reteplase, and tenecteplase. [12]

Fibrinolytic therapy is most effective when given within 3 hours after symptoms onset, although benefits have been seen when these drugs were given up to 12 hours. Ideally it should be initiated within 30 minutes of ASTEMI.

CONTRA INDICATIONS FOR THROMBOLYTIC THERAPHY

Absolute Contraindications :

- History of hemorrhagic stroke at any time
- Ischemic stroke within 3 months
- Intracranial neoplasm
- Active internal bleeding excluding menses
- Aortic dissection

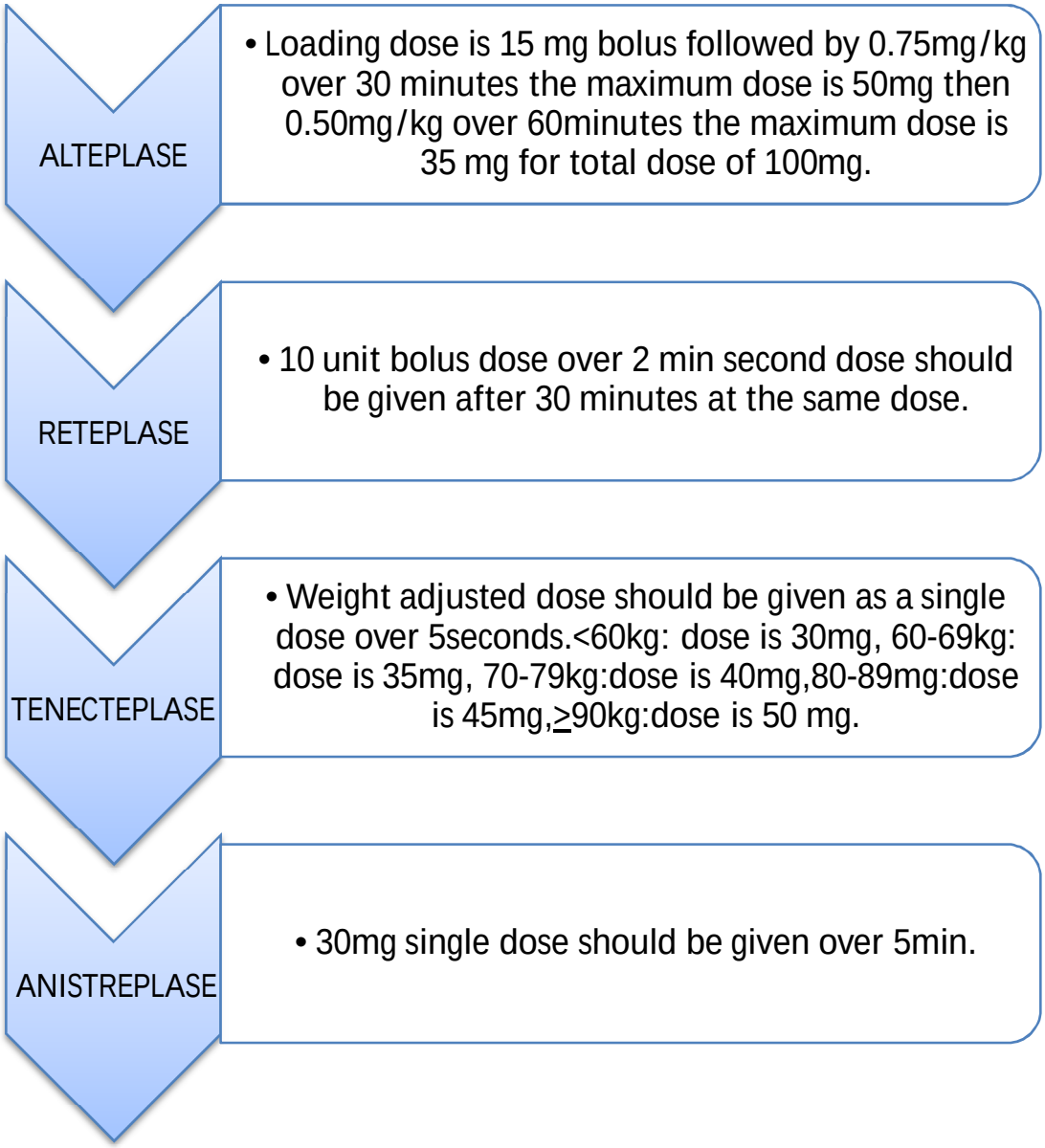
Relative contra indications :

- Blood pressure > 180 / 110 or < 90 / 60
- h/o chronic hypertension
- Bleeding diathesis
- Prolonged CPR / 10 min
- Recent trauma within 2 weeks
- Pregnancy
- Active peptic ulcer disease
- h/o prior streptokinase within 5 days to 2 years
- Major surgery less than 3 weeks
- Recent internal bleeding within 2 to 4 weeks.

STREPTOKINASE:

This agent is derived from hemolytic streptococci. Antibodies may produce after administration, so retreatment is usually avoided. It may allergic to patients can be seen in approximately in 5 % treated for the first time with a recent streptococcal infection. Allergic reactions respond to antihistamine. Less than 0.2 % of patients have a serious anaphylaxis. While on IV administration of streptokinase approximately 15% of patients will have hypotension, which usually responds to decreasing the rate of IV infusion and expansion of volume. It can produces a fibrinolytic state for up to 24 h. Streptokinase is less costly than other fibrinolytic agents.

OTHER FIBRINOLYTICS:



PERCUTANEOUS INTERVENTION:

Urgent percutaneous intervention is done in patients with ASTEMI when available in a timely manner. The door to balloon time is more than 90 min by an skilled operator. Indications for urgent percutaneous intervention are patients with shock, arrhythmias with patient requiring pacing methods or cardioversion, and elderly patient (age >75). [12]

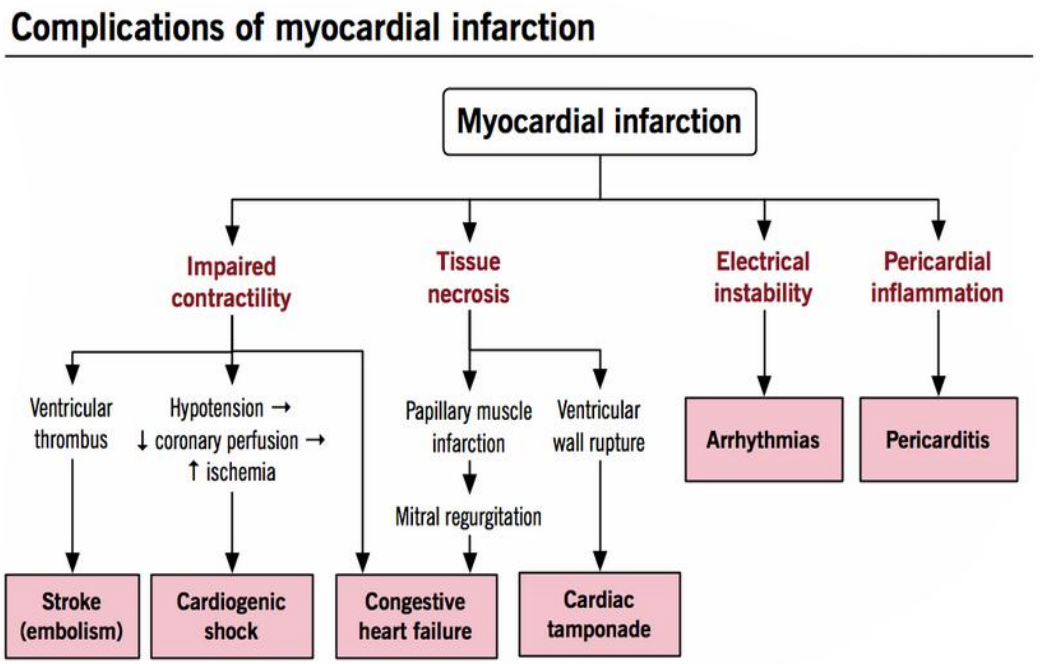
PROCEDURE:

Percutaneous intervention is the invasive procedures done by skilled operator. A catheter is passed through the femoral vessel then into the blocked coronary vessel in order to open blockages and restoration of the blood flow to the myocardium. PTCA means (Percutaneous transluminal coronary angioplasty) is the insertion of a catheter with a balloon attached with tip that is inflated to open the coronary vessel. A device known as a coronary stent made by a metal mesh can also be inserted in to the coronary vessel after angioplasty to keep the vessel open. Drug-eluting stents are used and they are coated with certain medications like sirolimus that prevent restenosis of the coronary vessel by reducing inflammation and thrombin formation. Coronary blockages can also be romoved by a procedure known as an atherectomy. Once the coronary vessel is opened with PTCA and brachytheraphy can be done by the radiation is delivered to the lesion. It helps to prevent narrowing or reblockage of vessel.

COMPLICATIONS:

Complications of percutaneous intervention are include hematoma from the arterial insertion site, decrease peripheral perfusion, arrhythmias and coronary spasm leads to MI bleeding in the retroperitoneal area, arrhythmias, coronary vessel spasm leads to MI, ARF, ischemic CVA and cardiac arrest. [12]

Sudden detorioration of previously stable patients should always think the "mechanical" complications of acute MI. As a group, these complications usually involve the tearing or rupture of affected tissue. These complications are less common in NSTEMI and USA.



FREE WALL RUPTURE:

It occurs in 10 % of AMI mortolity, usually 1 to 5 days after ASTEMI. The LV free wall rupture usually leads to tamponade and death in more than 90% of cases. Patients may feel sudden onset of severe tearing chest pain. They will be in shock and tachycardicrythm and may have onset of altered sensorium and restlessness. Increased JVP, diminished heart sounds, and pulsusparadoxus may be present. They are diagnosed by ECHO.

INTERVENTRICULAR SEPTAL RUPTURE:

Rupture of the IVS is more commonly detected clinically than rupture of the LV free wall, despite the fact that rupture of the LV free wall is more often detected in autopsy findings. The size of the defect and the degree of the shunt left to right can be related with prognosis of the ASTEMI. Clinically, IVS rupture presented with chest pain, breathlessness, and sudden hearing of pan systolic murmur. This can be associated with thrill felt in left lower sterna border. This is diagnosed by doppler study of echocardiography. An O2 step-up of more than 10 % from RA to RV samples is diagnostic. Rupture of the IVS is more common in patients with anterior wall MI and patients with extensive (3-vessel) disease. [12]

PAPILLARY MUSCLE RUPTURE:

Papillary muscle rupture occurs in approximately 1 percent of patients with AMI, is more common with inferior MI, and usually occurs 3 to 5 days after AMI. In contrast to rupture of the IVS, papillary muscle rupture often occurs with a small to medium sized acute MI. Patients may have relatively limited CAD. Patients present with acute onset of breathlessness, increasing degree of CHF, and a new pan systolic murmur consistent with mitral regurgitation. The posteromedial papillary muscle is most commonly ruptured, because it gets blood supply from one coronary vessel, usually the right coronary vessel. Echocardiography can differentiate rupture of a portion of the papillary muscle from other causes leads to MR. Immediate surgical intervention is the treatment of choice for this acute MR.

PERICARDITIS:

In ASTEMI 10 to 20 % of patients will get pericardial inflammation. It is mainly occurs in transmural infarction. It usually occurs within two to four days after AMI. It is clinically diagnosed by hearing pericardial friction rubs. This is mostly seen in RV infarction or inferior wall MI, because the RV lies immediately beneath the chest wall. Sometimes,the pain of pericarditis can be confused with that of post infarction angina. Specific nature of the pain is increased while on deep inspiration and relieved by sitting and leaning forward. Pericardial

effusions are associated with pericarditis, it also occurs in the absence of pericarditis. The resorption rate of post MI pericardial effusions is very slow, usually taking many months. Post MI syndrome or “dressler syndrome” occurs 2 to 10 weeks after ASTEMI and patient presented with high fever, left chest pain and pleuropericarditis. [12]

RV MI:

Isolated RV infarction is very rare, and it is usually seen as a complication of an IWMI. The RV most commonly receives its blood supply from the right coronary vessel. In patients with left dominant systems, the blood supply may come from the Lcx. The anterior portion of the RV is supplied by branches of the left anterior diagonal vessel. Approximately 30% of IWMI involve the RV. The presence of RV infarction is associated with a significant increase in mortality and cardiovascular complications. RV infarction can be diagnosed by the presence of ST-segment elevation in the precordial V_{4R} lead in the setting of an IWMI. The presence of increased JVP or decreased BP in response to NTG is also suggestive.

The most serious complication of RV infarction is shock. The severity of the hemodynamic derangement in the setting of RV infarction is related to the extent of RV dysfunction, the interaction between the ventricles (the RV and LV share the IVS), and the interaction between the pericardium and the RV. RV infarction results in a reduction in RV end-

systolic pressure, LV end-diastolic size, cardiac output, and aortic pressure as the RV becomes more of a passive conduit to blood flow. LV contraction causes bulging of the IVS into the RV, with resultant ejection of blood into the pulmonary circulation. As a result, RV infarction with concurrent LV infarction has a particularly devastating effect on hemodynamic function. Fluid balance and maintenance of adequate preload are critical in the treatment of RV infarction.

OTHER COMPLICATIONS:

Other complications of ASTEMI that occur but are not usually seen in the emergency department include LV thrombus formation, thrombosis of venous and arterial system, PE postinfarction angina, and infarct extension. [12]

CARDIAC BIOMARKERS:

Initially, WHO had ascribed equal weight to ECG and cardiac biomarkers for diagnosis of acute MI. However, increasing precision of CK-MB using assays and increasing value of cardiac troponin assays has now established the use of cardiac biomarkers as main marker of myocardial necrosis. Other biomarkers are SGOT and LDH.

Serum cardiac biomarkers of acute ASTEMI are proteins or macro molecules released from the cardiac cells (myocytes) undergoing necrosis. 10–14 Markers vary in terms of their molecular weight, cellular

location, solubility, plasma concentration, clearance, and ability to be detected accurately in serum with rapid immunochemical techniques. In general, serial measurements are more sensitive and accurate than initial single measurements, and serial sampling every 2 to 3 hours is a validated practice for the rapid exclusion of AMI in acute chest pain patients.

Cardiac biomarkers can be currently described under the following head:

- Estabilished cardiac biomarkers
- Emerging biomarkers
- Developing biomarkers

ESTABILISHED BIOMARKERS:

MYOGLOBIN:

Myoglobin leaks from necrotic myocardial tissue far more rapidly than AST, CK and LDH. Early rise and high sensitivity are two properties that have led to the adoption of myoglobin as part of multimarkerstratagies. There is also a greater availability of assays for myoglobin. However, in view of its very poor specificity, myoglobin appears to be less acceptable than delayed troponin testing. Rapid release kinetics also make myoglobin usefull for non invasive monitoring of reperfusion in patients with ASTEMI.

TROPONIN I and T:

Troponin is at present the preferred biomarker for diagnosis of AMI. Since it has a higher sensitivity for detecting myocardial injury than CK-MB and it also provides useful information about prognosis. Blood samples for measurement of troponin should be drawn on first assessment and six to nine hours later. Troponin is started to rise two to four hours of acute MI and remains elevated up to seven days to fourteen days. Normal level is less than 0.4ng per ml. Although troponin T and troponin I assays usually provide equivalent clinical information, standardization is a problem with troponin I since large number of commercially available assays are available. Each assay has its specific analytical characteristics and it is important that clinicians should be conversant with the s of the particular assay for correct interpretation. Most of the current assays use a cut-off in clinical practice in the range of 0.03 t0 0.08.

CREATINE KINASE-MB

Creatine kinase-MB(CK-MB) is a cytosolic carrier protein for high energy phosphates and for a long time, it has been the gold standard for AMI diagnosis. Its elevation occurs four to eight hours, after MI and peaking occurs at eighteen to twenty four hours. The sensitivity of CK-MB for ASTEMI is only 50% when measured early at the time of presentation. Its sensitivity and specificity can be increased by serial testing. Serial testing of CK-MB also enables the physician to detect early

recurrent infarction. Myocardial damage is likely if the False positive results can be seen in muscloskeletal injury, post operative conditions, RF and peripartam period.

Electrophoretic methods were used to detect isoenzymes originally; Peak level of markers of necrosis and area under time-release curve of CK-MB from repetitive serial sampling has been used to estimate infarct size. After the introduction of cardiac troponin assays in clinical practice,CK-MB has had a progressive diminution in its role in diagnosis and prognostication of ACS

EMERGING BIOMARKERS:

- BNP and N-terminal pro BNP
- C-reactive protein

DEVELOPING BIOMARKERS:

- Low sensitivity in the first 4-6 hours after onset of chest pain
- Poor markers of ischemia in absence of myocardial necrosis
- Affected by inflammation or injury to other body systems

1. Myeloperoxidase
2. Soluble CD40 ligand
3. Matrix metalloproteinase-9
4. Fatty acid binding proteins

5. Ischemia modified albumin
6. Free fatty acid unbound to albumin
7. Placental growth factor
8. PAPA-pregnancy-associated plasma protein A

ECHO

Echocardiography is the most useful and easily available modality to diagnose ASTEMI. MI results in regional LV (LV) wall motion abnormality (RWMA) and it present before the symptoms and electrocardiogram abnormalities in onset. If RWMA is not present during an episode of chest pain, diagnosis of ASTEMI is unlikely and hence it is recommended as a diagnostic modality in patients with non diagnosticeletrocardiogram, especially during pain or shortly after it ceases. Another common problem is distinguishing RWMA due ischemic changes from old infarction. Echocardiography also excludes other causes of chest pain like cardiac tamponade, aortic dissection and PE. Newer advancements like strain imaging by tissue Doppler technique hold the potential to detect wall motion abnormalities earlier than currently used 2D imaging. Assessment of systolic-diastolic LV function by echocardiography is a strong-determinant of prognosis. [12]

ERYTHROCYTE SEDIMENTATION RATE:

The erythrocyte sedimentationrate is done by Westergren method or Wintrobes method. Itisthe commonest test and it is used for measuring the inflammation rate atwhichErythrocytesedimentation rate done ina onehour.

EdmundBiernacki invented this test in 1897 and this test called asbiernacki reaction. The SwedishpathologistsRobertSannoFahraeus invented this test in 1918 along withAlfVilhelmAlbertssonWestergren,declaredthis method andarereferredasWestergrenmethod which contains sodium citrate-coagulated specimens. [14]

Whenaninflammation occurs, that leads to increase fibrinogen level in the serum that causes RBCs to stick to each other. Theerythrocytesformstackscalledrouleaux formation, whichsettle faster. [2]

NORMAL VALUE: ESR

Sex	Male	Female
50 yrs. old or less	15mm/h	20mm/hr
Over 50 yrs old	20mm/hr	30mm/hr

EA AND ADHESIVENESSTEST (EAAT):

EA/ adhesiveness test (EAAT) is asimplebedsideslide test.Itisa very feasible,cost effective,fastmethodofdirectly visualizingtheEAstatus.Itisa usefulbiomarker todetectinternalinflammationin individuals with atherothrombotic risk factors. [9]

1. Thetest has the potential toassess the risk ofacute MI

2. It can beused to assess the prognosis in patient with

ASTEMI.

Methodology

METHODOLOGY

MATERIALS AND METHODS:

Study design: Cohort study design

Place of study: Government Royapettah Hospital,

Collaborating Department:

Department of Cardiology, Government RoyapettahHospital, KilpaukMedical College.

Period of study: May 2014 to Sep 2014

Sample size: 50

Study population (subjects): Patients admitted in ICCU with ASTEMI at GRH during study period.

Inclusion criteria:

1. Patients admitted in ICCU with acute ASTEMI
2. Left side chest pain more than thirty minutes, within six hours ofonset
3. ST segment elevation more than one millimeter in 2 leads in ECG

Exclusion criteria: 1. Recent MI,

2. Sepsis,

3. Infections-any bacterial infection

4. Neoplasms

5. Liver failure,ESRD

6. DVT,

7. Patient on antiplatelet drugs statins

ASTEMI patientsofallagegroupssattisfy theinclusioncriteriaandadmitted tothe ICCUof Government Royapettah Hospital, Kilpauk medical college wereincludedinthe study.Informedconsentwastakenfromthe patients.Past history of the patient wasaskedandroutine examination like both general and systemic examinationwasdone.Completehistory was asked includingRFofthe coronary vessel diseasewere recorded.Blood wasdrawnedfromantecubitalvein at the time of admission. The ASTEMIsubjectsweretreatedbythe standardtreatmentguidelinesofourhospital.Thebloodsampleswere and drawned from the subjects and immediately sendtothelaboratoryandESR (erythrocyte sedimentation rate)by Westergren'smethodandslidetest(EAAT)were performedalongwith

otherinvestigations.

1. Fifty subjects of acute ASTEMI admitted to the Department of Cardiology, Government Royapettahhospital,onset within six hours, chest pain duration is more than 30 min, and two or more contiguous leads with ST segment elevation .

2. Informed consent was taken from the subjects patients with ASTEMI

3. History was asked from the patients, and routine general examination and syASTEMIc examination were done. Risk factors of the CAD were also recorded.

4. Anticoagulatedbloodwasinstilledover the simpleslideandbloodsmears were stained by leishman stain andobserved under400X oil immersion field andgraded into four

SLIDE PREPARATION:

Onedropof citrated anticoagulant mixed with threedropsofblood and poured over the glass slide.Thiscitrated anticoagulantbloodwaspouredona slide and positioned the slide at 45 degree. Wait for 10 seconds for blood ran from above downwards and make a thin smear. The lowest part of the slide was wiped by absorbent

tissue paper. The slides were kept at room temperature and dried by room temperature itself.

Thesize of blood drop andthe angle of slides were constant in all subjects with ASTEMI. Staining was done by 5 to 6 drops of Leishman stain and wait for two minutes. Distilled water gently instilled over the slide double the amount of leishman stain and wait for eight minutes. Finally, washed under the tap water and dried in room temperature. Oil immersion field was used for the assess the grades of EA

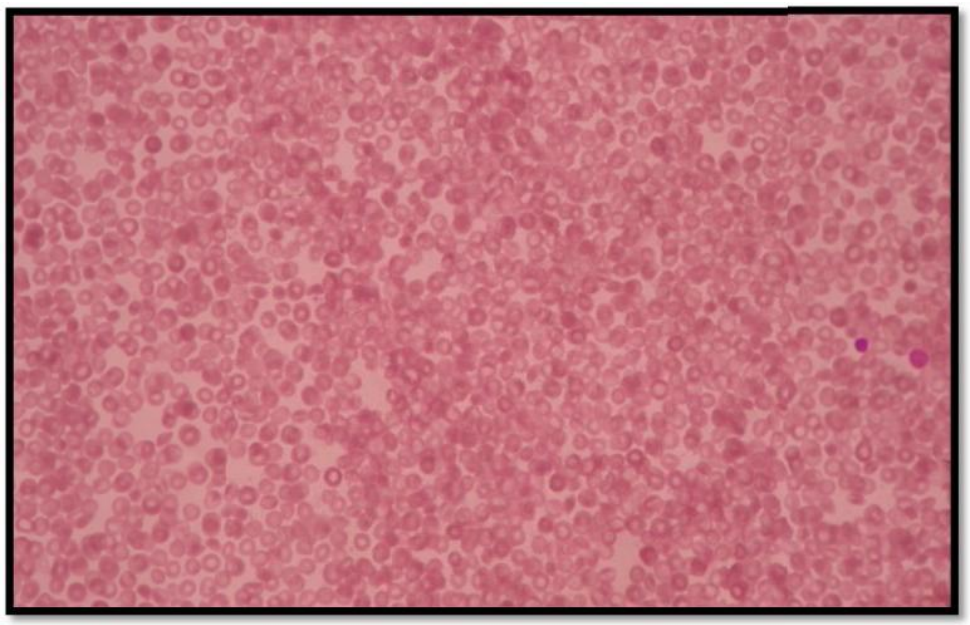
ANALYSIS OF THE SLIDES

The slides were observed under oil immersion fields in microscope with fourty× (four hundred×)magnification. The slides were fully examined and assigned grades of red cell aggregation in to mild (A),moderate(B),Severe (C) and very severe (D) by the investigator.

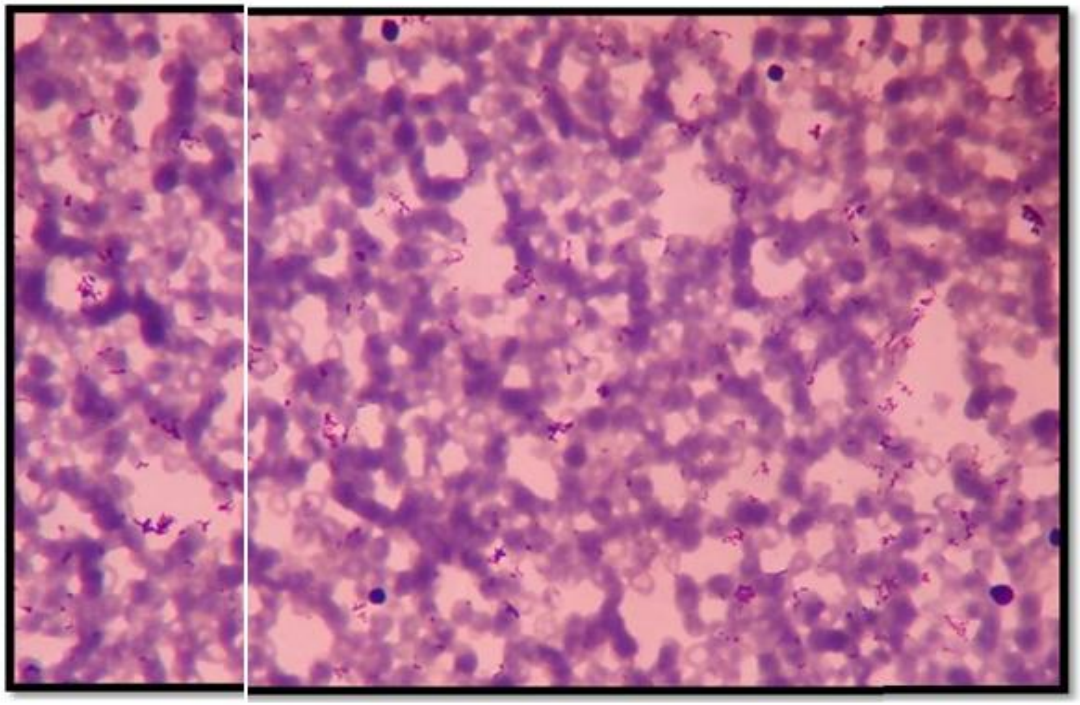
CRITERIA FOR GRADING RED AGGREGATION

GRADE

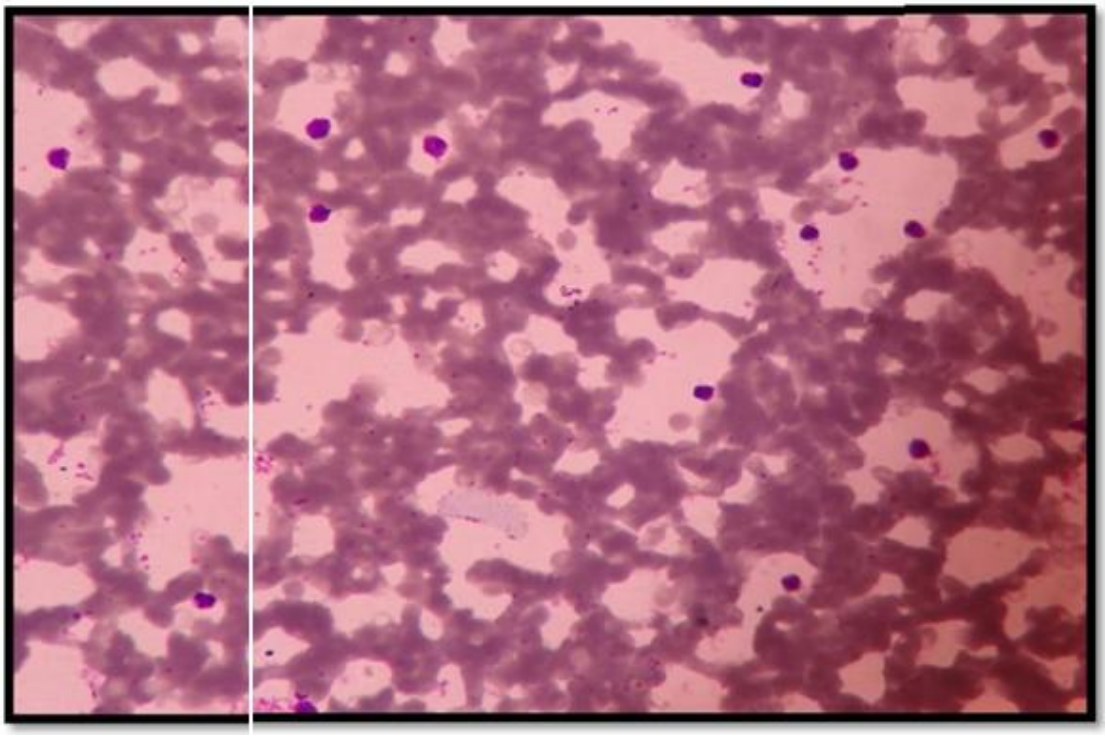
A:RBCsarediscretewithuniformdistributionthroughoutwithno clear areas in between. Picture



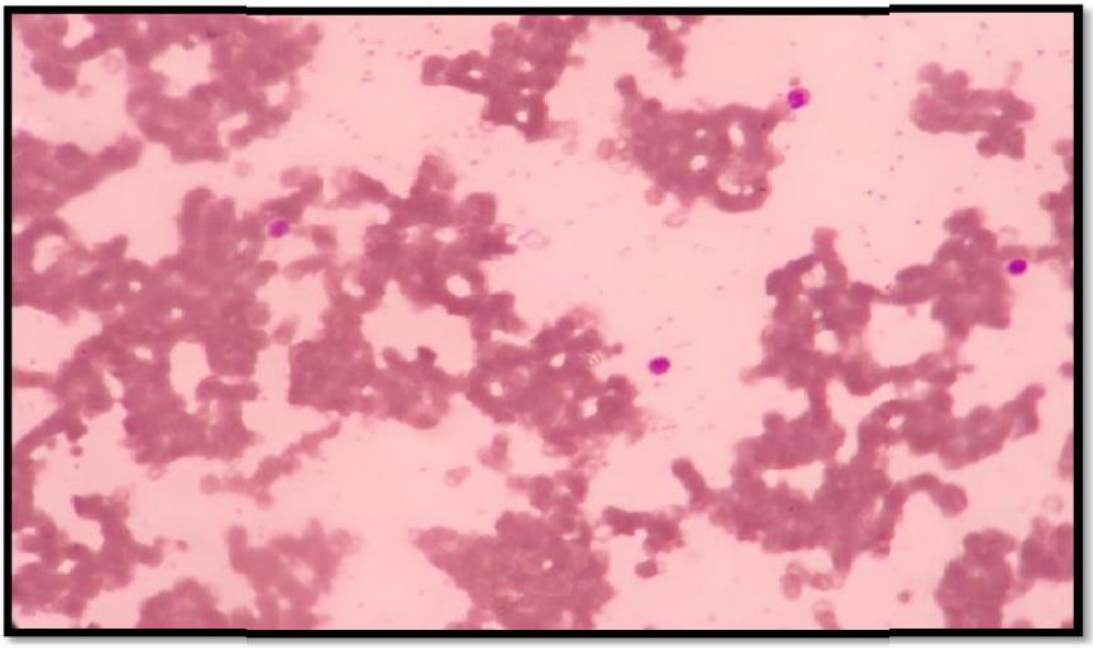
GradeB:RBCsareaggregatedinsomeareasoftheslidewith areas of small clear spaces. picture 11



GradeC: Variablesizesoferythrocytes aggregatesinallthe areas ofslidewithsmall clear spaces. Picture



GradeD: LargethickRBCaggregateswithrounded/clearbordersandlarge clearspaces.Picture



The grades of red cell aggregation were observed by standard criteria. The grades of EA are compared with prognosis of the ASTEMI patients with one week follow up.

PROGNOSIS:

Good prognosiswasdocumented in patients with complete recovery.

Bad prognosiswas documented in patients

- Recurrent chestpain orAngina
- Re- infarction
- LV failure
- Cardiogenicshock
- Cardiac arrhythmias,VT,VF,AF
- Mortality from cardiac causes
- Mortality from non cardiac causes

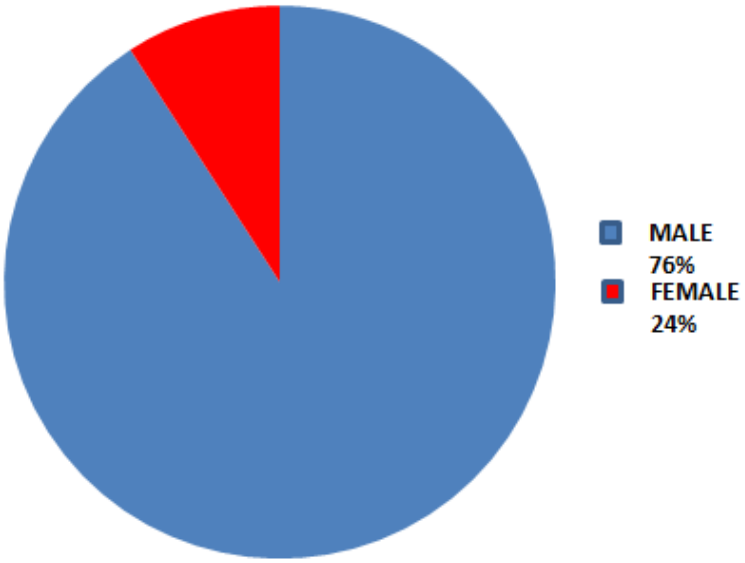
Observation & Results

OBSERVATION AND RESULTS

Out of 50 patients males were 38 females were 12. In out of 100 percentage males were 76% and females were 24%.

		Freq	%	Valid %	Cumulative %
Valid	Male	38	76.0	76.0	76.0
	Female	12	24.0	24.0	100.0
	Total	50	100.0	100.0	

Table: 1 Sex difference of the patients



Picture: 22 - sex ratio of the ASTEMI patients

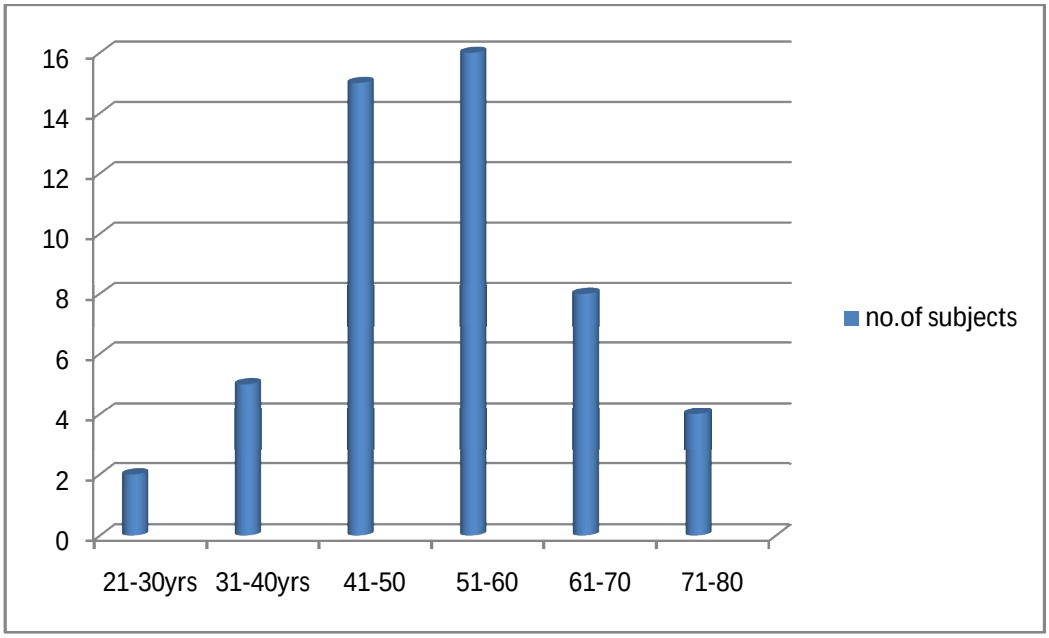
Table: 2 Age and sex distribution of ASTEMI patients

Sex	Age in yrs
Males	28-77yrs
Females	44- 75yrs
Overall	28-77yrs

		Frequency	%	Valid %	Cumulative %
Valid	21-30 yrs	2	4.0	4.0	4.0
	31-40 yrs	5	10.0	10.0	14.0
	41-50 yrs	15	30.0	30.0	44.0

51-60 yrs	16	32.0	32.0	76.0
61-70 yrs	8	16.0	16.0	92.0
71-80	4	8.0	8.0	100.0
Total	50	100.0	100.0	

Table: 3 age group with frequency



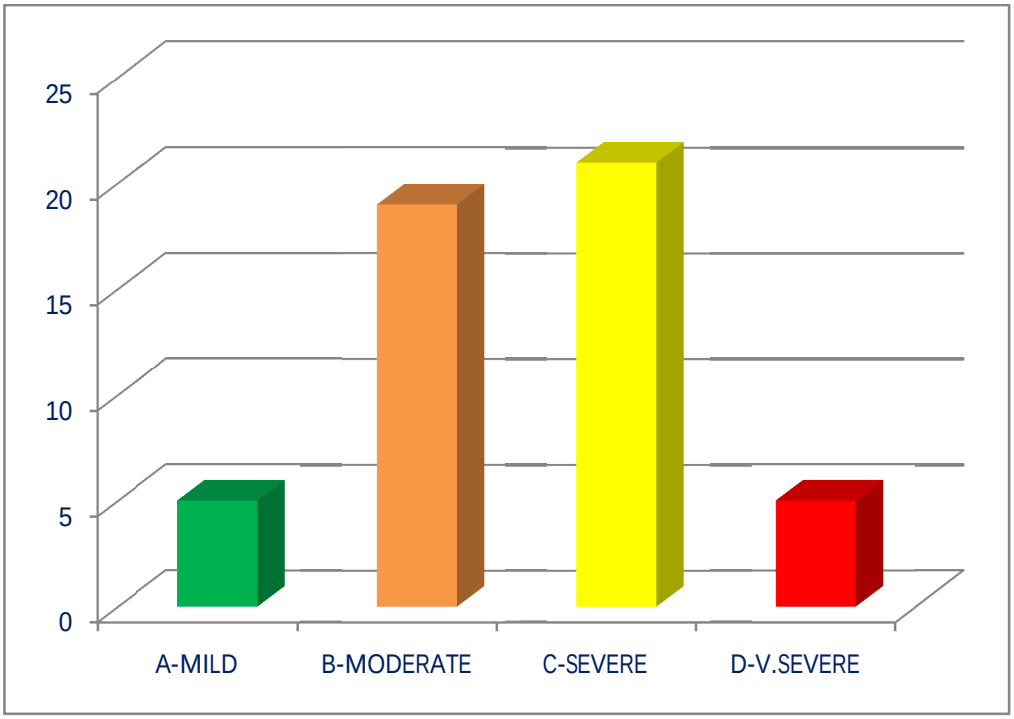
Picture: 23 - Age distribution of ASTEMI patients

Age group more commonly came with acute MI are 51-60 years, next age group are 41-50years then 61-70 years.

EA:

		Freq	%	Valid %	Cumulative %
Valid	A	5	10.0	10.0	10.0
	B	19	38.0	38.0	48.0
	C	21	42.0	42.0	90.0
	D	5	10.0	10.0	100.0
	Total	50	100.0	100.0	

Table: 4 Percentage of EA in ASTEMI patients



Picture: 24 - % of EA in ASTEMI

Out of fifty patients mild aggregation Grade A are observed in 5(10%) patients, Grade B EA observed in 19(38%) patients. Grade C aggregation observed in 21(42%) patients. Grade D aggregation observed in 5(10%) patients.

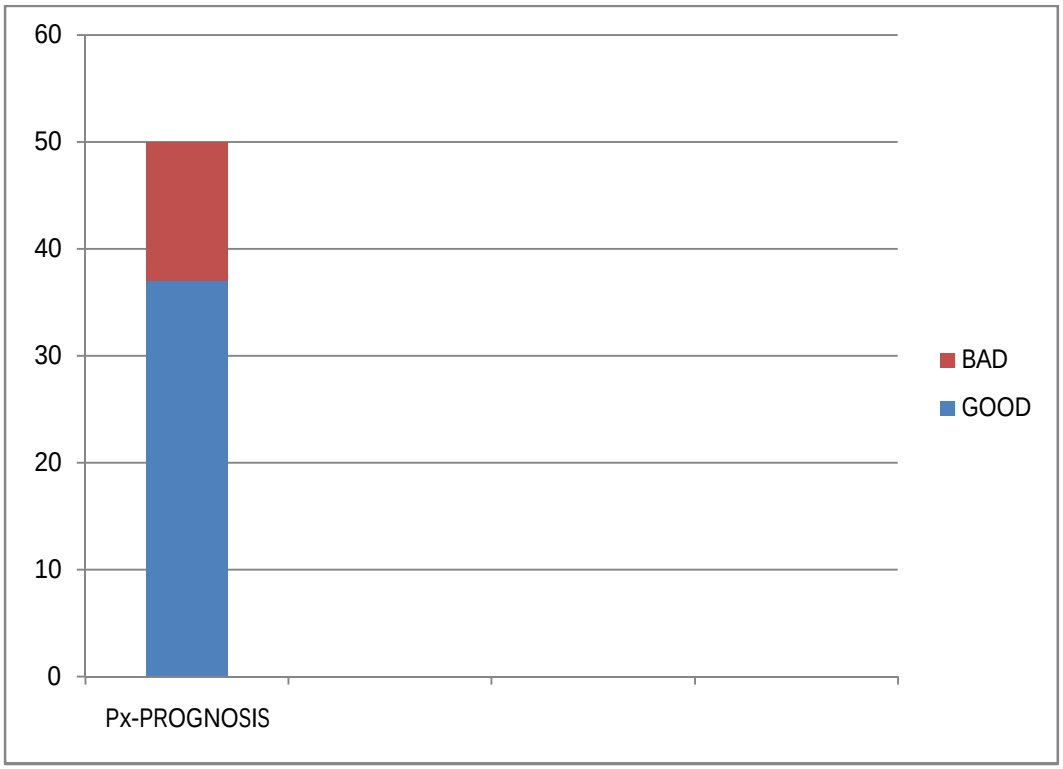
EA grades with prognosis of the patient

		Frequency	%	Valid %	Cumulative %
Valid	Good	37	74.0	74.0	74.0
	Bad	13	26.0	26.0	100.0
	Total	50	100.0	100.0	

Table: 5 Good and Bad Px in total number of cases

Out of fifty patients 12 patients were female and 7(58.3%) patients had good prognosis and 5(41.7%) patients had bad prognosis.38(78.9%) patients were male among this 8(21.1%) patients had bad prognosis.

Out of fifty patients with ASTEMI 5 showed Grade A RBCs aggregation, all of them had Good prognosis (100%).



Picture: 25 - Good and Bad Px in total number of patients

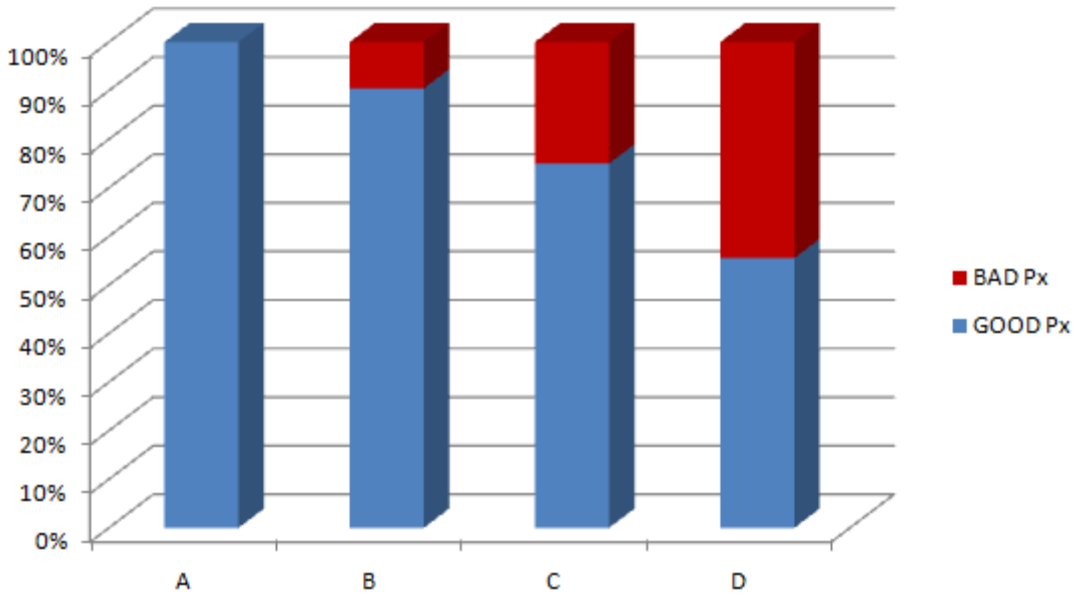
Out of fifty patients 13 patients ASTEMI had Bad prognosis within one week period of followup.37 patients had Good prognosis.

In patients Grade B RBCs aggregation was observed, out of this 17(89.5%) had good prognosis and 2(10.5%) patients had bad prognosis. Grade C RBCs aggregation observed in 21 patients, out of this 14 patients (66.7%) had good prognosis and 7patients (33.3%) had bad prognosis. Grade D RBCs aggregation observed in 5 patients, out of this 4(80%) had good prognosis and 1(20%) had good prognosis.

			Prognosis within 1 week period		Total
			Good	Bad	
EA grade	Mild	Count	5	0	5
		% within EA grade	100.0%	.0%	100.0%
		% within Prognosis within 1 week period	13.5%	.0%	10.0%
	Moderate	Count	17	2	19
		% within EA grade	89.5%	10.5%	100.0%
		% within Prognosis within 1 week period	45.9%	15.4%	38.0%
	Severe	Count	14	7	21
		% within EA grade	66.7%	33.3%	100.0%
		% within Prognosis within 1 week period	37.8%	53.8%	42.0%
	very Severe	Count	1	4	5
		% within EA grade	20.0%	80.0%	100.0%
		% within Prognosis within 1 week period	2.7%	30.8%	10.0%
Total		Count	37	13	50
		% within EA grade	74.0%	26.0%	100.0%
		% within Prognosis within 1 week period	100.0%	100.0%	100.0%

Table: 6 Grades of EA Vs prognosis

Picture: 26 - EA grades Vs prognosis with in 1 week



ASTEMI patients with Grades C and D were significantly associated with bad prognosis, similarly Grades A and B were significantly associated with good prognosis. It was done by Chi-square Test and the P value is observed P=0.006.This P value is <0.05, hence it is statistically significant.

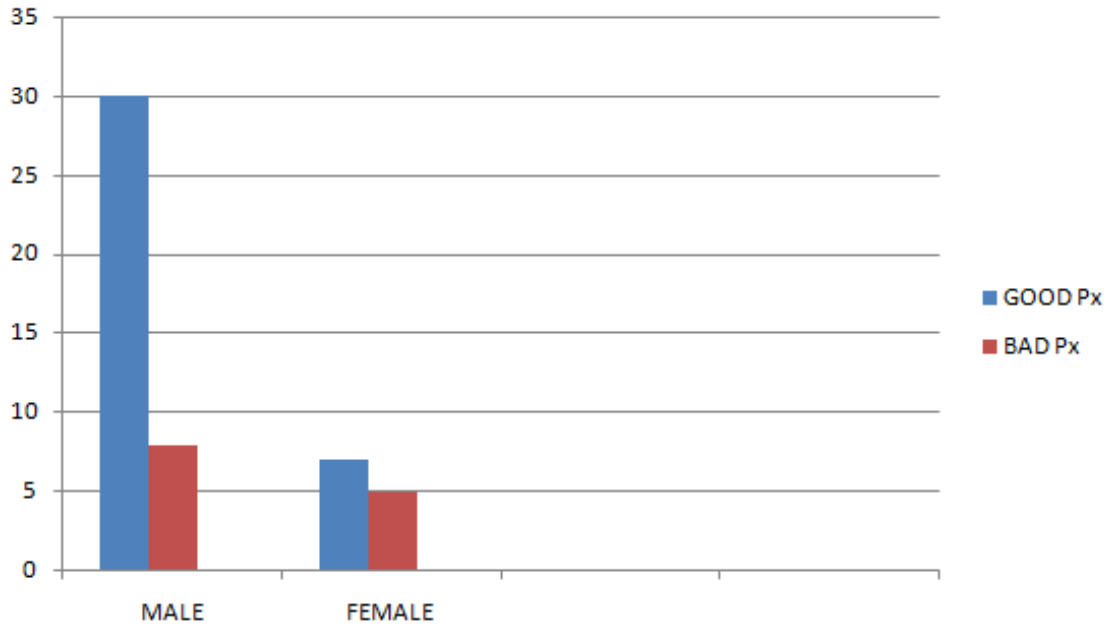
	Value	df	Significance p
Pearson Chi-Square	12.286(a)	3	.006
Likelihood Ratio	12.781	3	.005
Linear-by-Linear Association	10.648	1	.001
N of Valid Cases	50		

Table:7 Chi-square test

Sex ratio Vs Prognosis:

			Prognosis within 1 week period		Total
			Good	**Bad**	
Sex	Male	Count	30	8	38
		% within Sex	78.9%	21.1%	100.0%
		% within Prognosis within 1 week period	81.1%	61.5%	76.0%
	Female	Count	7	5	12
		% within Sex	58.3%	41.7%	100.0%
		% within Prognosis within 1 week period	18.9%	38.5%	24.0%
Total		Count	37	13	50
		% within Sex	74.0%	26.0%	100.0%
		% within Prognosis within 1 week period	100.%	100.0%	100.0%

Table:8 Prognosis in males Vs Females



Picture: 27 - Sex ratio Vs Px

OTHER VARIABLES COMPARED WITH GRADES OF EA

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
SGOT	Between Groups	1050.657	3	350.219	1.466	.236
	Within Groups	10988.623	46	238.883		
	Total	12039.280	49			
Cardiac Enzymes - CPK – MB	Between Groups	311.461	3	103.820	.980	.410
	Within Groups	4872.859	46	105.932		
	Total	5184.320	49			
Cardiac Enzymes - TROP - T	Between Groups	1.949	3	.650	1.627	.196
	Within Groups	18.371	46	.399		
	Total	20.320	49			
TG	Between Groups	644.964	3	214.988	1.067	.373
	Within Groups	9272.256	46	201.571		
	Total	9917.220	49			
LDL	Between Groups	689.788	3	229.929	1.957	.134
	Within Groups	5403.332	46	117.464		
	Total	6093.120	49			
HDL	Between Groups	133.507	3	44.502	1.450	.241
	Within Groups	1412.173	46	30.699		
	Total	1545.680	49			
Total Cholesterol	Between Groups	970.333	3	323.444	1.624	.197
	Within Groups	9160.547	46	199.142		
	Total	10130.880	49			
ESR	Between Groups	75.925	3	25.308	1.512	.224
	Within Groups	770.075	46	16.741		
	Total	846.000	49			
Leucocyte Count	Between Groups	1891524.311	3	630508.104	.377	.770
	Within Groups	76878275.689	46	1671266.863		
	Total	78769800.000	49			
	Between Groups	523.250	3	174.417	1.285	.291
	Within Groups	6243.870	46	135.736		
	Total	6767.120	49			

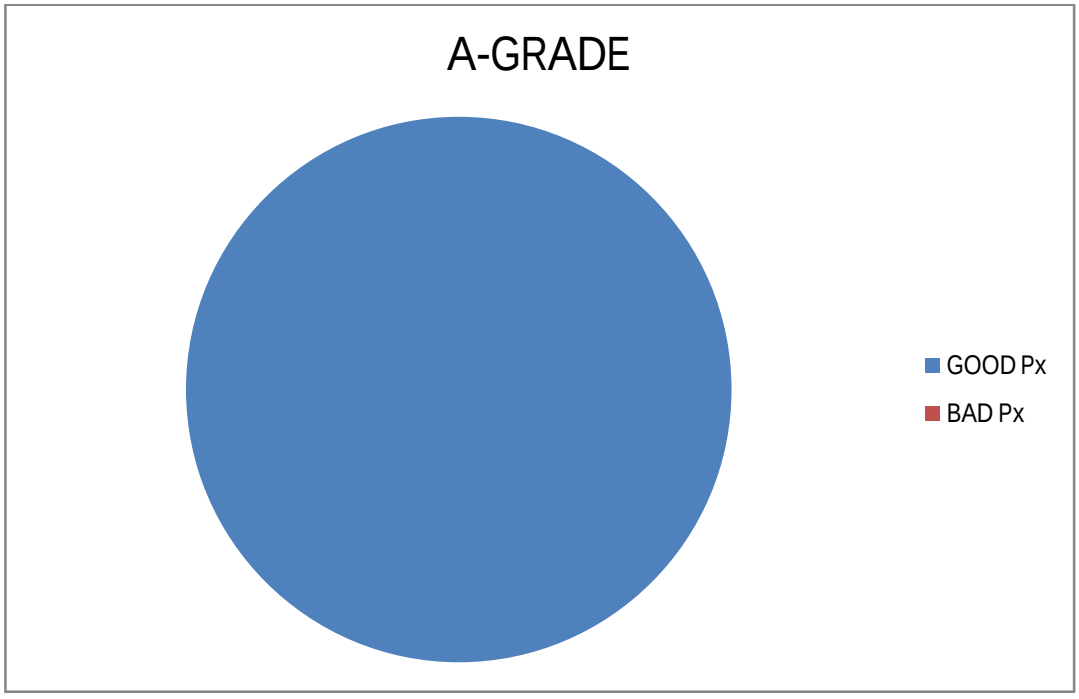
Table:9Anova Other variables Vs prognosis

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	Df	Sig. (2-tailed	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
ESR	Equal variances assumed	.046	.830	-1.313	48	.195	-1.75	1.330	-4.420	.928
	Equal variances not assumed			-1.250	19.341	.226	-1.75	1.398	-4.668	1.175
Leucocyte Count	Equal variances assumed	.656	.422	-.702	48	.486	-288.36	410.918	-1114.564	537.849
	Equal variances not assumed			-.779	25.985	.443	-288.36	370.080	-1049.089	472.374
SGOT	Equal variances assumed	2.156	.149	-1.556	48	.126	-7.75	4.982	-17.768	2.267
	Equal variances not assumed			-1.698	25.011	.102	-7.75	4.566	-17.154	1.653
Cardiac Enzymes - CPK – MB	Equal variances assumed	3.021	.089	-2.460	48	.018	-7.77	3.158	-14.116	-1.418
	Equal variances not assumed			-2.164	17.230	.045	-7.77	3.589	-15.331	-.204

Cardiac Enzymes - TROP - T	Equal variances assumed	1.248	.270	-.487	48	.629	-.102	.2093	-.5226	.3189
	Equal variances not assumed			-.446	18.269	.661	-.102	.2282	-.5807	.3770
TG	Equal variances assumed	1.664	.203	-2.074	48	.143	-9.21	4.440	-18.134	-.281
	Equal variances not assumed			-2.446	29.939	.121	-9.21	3.764	-16.895	-1.521
LDL	Equal variances assumed	.525	.472	.032	48	.975	.12	3.633	-7.187	7.420
	Equal variances not assumed			.031	19.581	.976	.12	3.788	-7.797	8.030
HDL	Equal variances assumed	.000	.984	-1.150	48	.256	-2.07	1.805	-5.704	1.554
	Equal variances not assumed			-1.159	21.351	.259	-2.07	1.791	-5.795	1.646
Total Cholesterol	Equal variances assumed	4.731	.035	-2.586	48	.113	-11.35	4.388	-20.170	-2.524
	Equal variances not assumed			-3.093	30.981	.104	-11.35	3.669	-18.830	-3.864

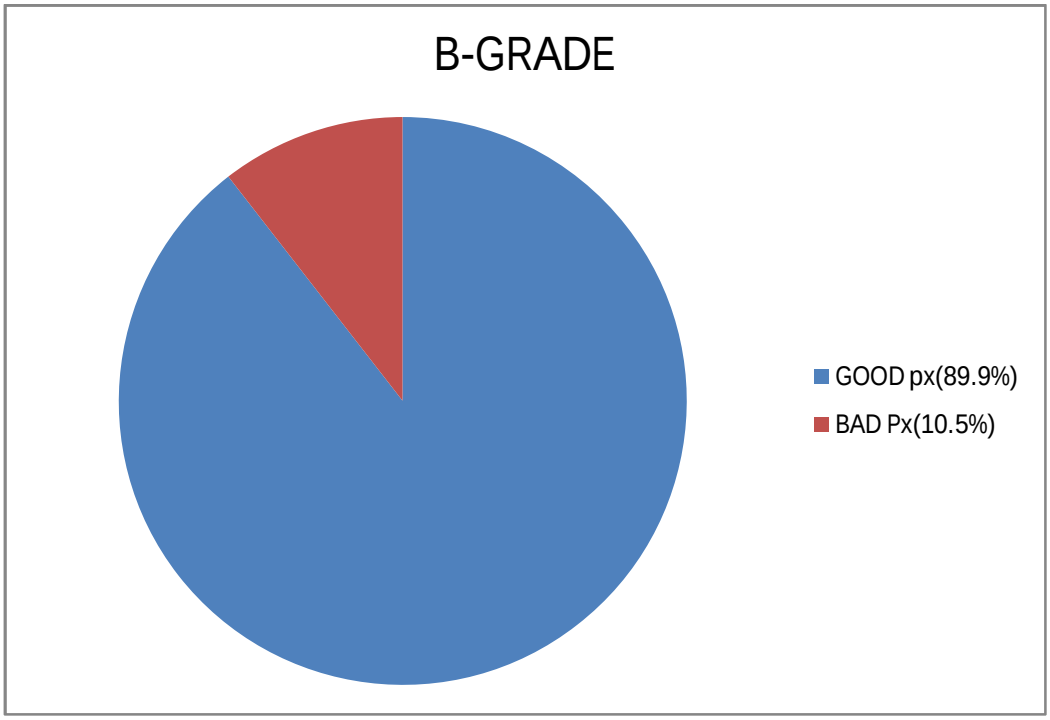
Table10 other variables with prognosis P values

Other variables, ESR, leucocyte count, SGOT, cardiac enzymes and lipid profile were corrfelated with prognosis of the ASTEMI patients.All are not statistically significant except cardiac enzymes CPK-MB.



Picture: 28 - EA A grade Vs Px

Picture: - 29 Percentage of EA B Grade with Good and Bad Px



Picture: 30 - Percentage of C GRADE with Good and Bad Px

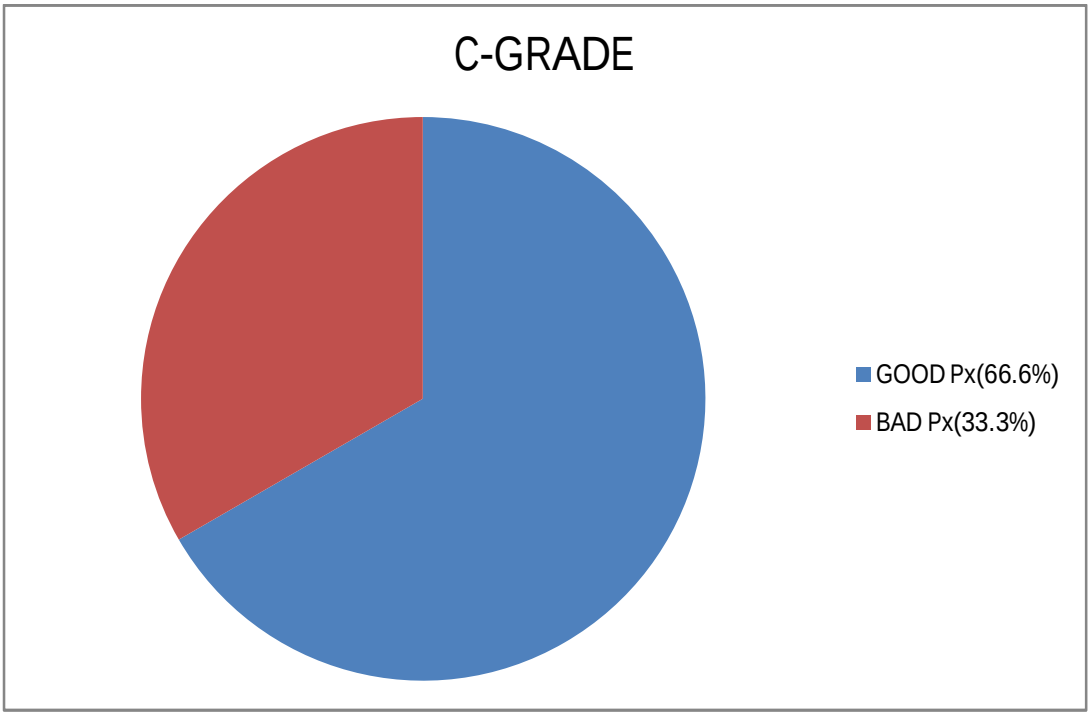
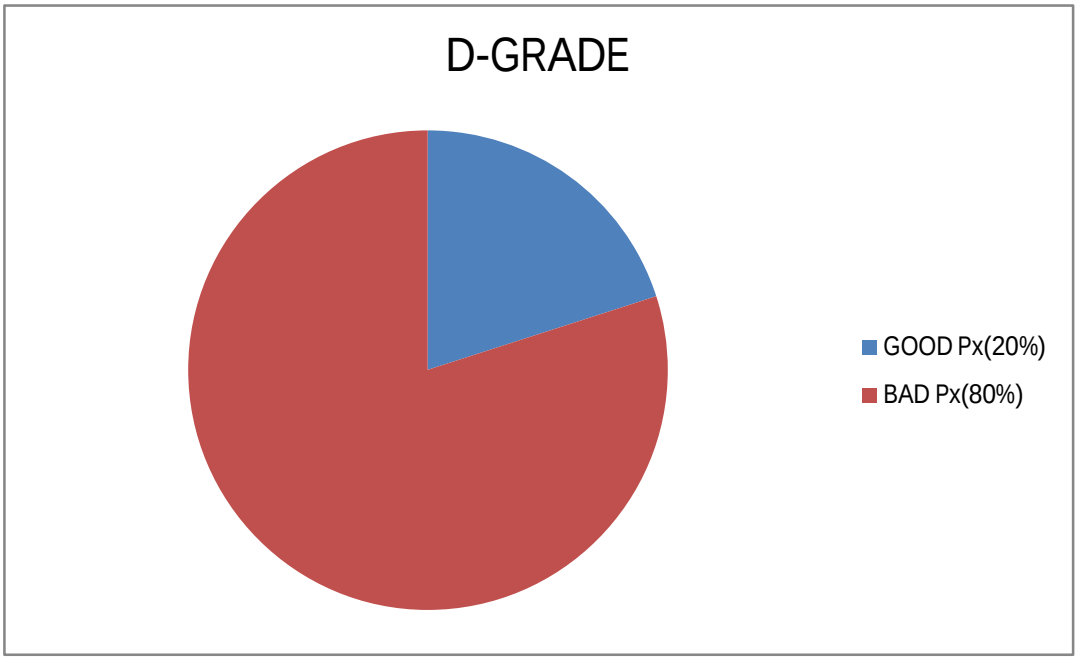
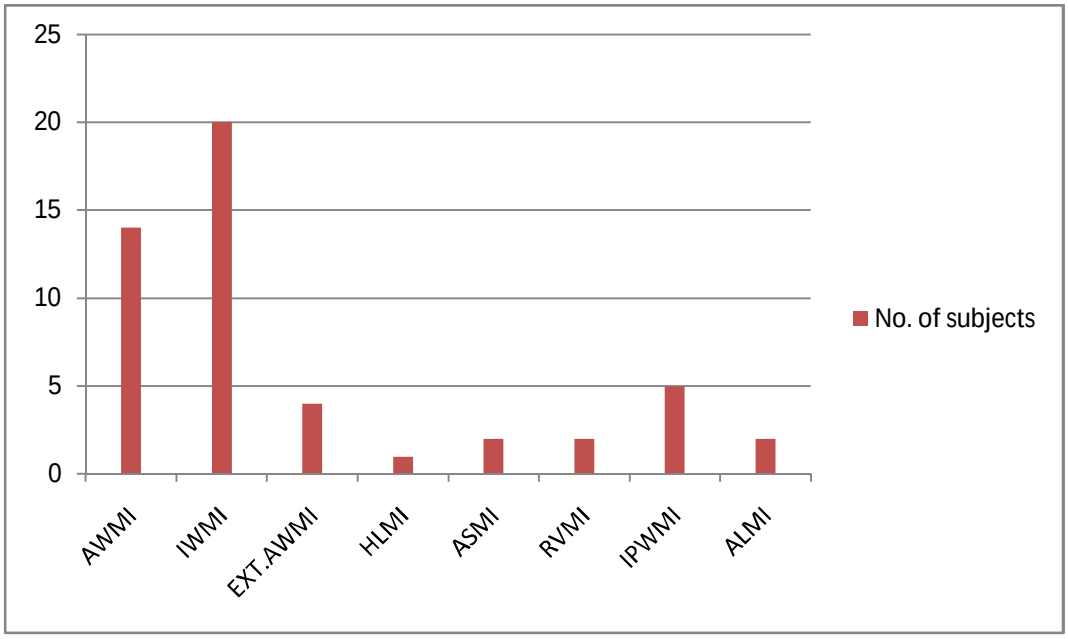


Figure: 31 - Percentage of D GRADE with Good and Bad Px



		Frequency	%	Valid %	Cumulative %
Valid	ALMI	2	4.0	4.0	4.0
	ASMI	2	4.0	4.0	8.0
	AWMI	14	28.0	28.0	36.0
	EXT	4	8.0	8.0	44.0
	HLMI	1	2.0	2.0	46.0
	IPW MI	5	10.0	10.0	56.0
	IWMI	20	40.0	40.0	96.0
	RVMI	2	4.0	4.0	100.0
	Total	50	100.0	100.0	

Table: 11 ECG frequency in subjects with ASTEMI



Picture: 32 - ECG frequency in subjects

Inferior wall MI is more frequency than other ASTEMI, second common frequency is Anterior wall MI, less common frequency is RV MI and High lateral MI.

COMPLICATIONS:

		Frequency	%	Valid %	Cumulative %
Valid	Nil	37	74.0	74.0	74.0
	Died	6	12.0	12.0	86.0
	Arrythmia	4	8.0	8.0	94.0
	LVF	2	4.0	4.0	98.0
	Cardiogenic Shock	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

Table: 12- Frequancy of complications in subjects

Out of this fifty patients 37(74%) patients had good prognosis, 4(8%) patients had arrhythmia, 2 patients (4%) had LVF, one patient went to cardiogenic shock. Six (12%) patients died from ASTEMI.

Discussion

DISCUSSION

Acute MIis a major cause of morbidity and mortality worldwide.Atherosclerosisi m p l y p u t i s a chronicinflammationofthevessel wall. Many researchershaveperformed numerous studiesoverinflammatory markers in a question to findreliableindicatorfor CHD riskand progression. [16,17,18]In Acute MI, numerousinflammatorymarkershave been studied and havebeen linked to poor prognosis.[19-27]

StudiesweredonetoestablishthatEAformationwashigh inpatientswithCHD.Itis well knownthatthe EAis determined by forcesactingagainst eachother,repulsionofnegatively charged cells,cell-to-celladhesionduetoplasmaproteinsandthe force generated byblood flow.[28-32] EA is increased in various inflammatory conditionssuchasAMI,acute stroke, sepsis etc. [33,34]and it affects theoutcomein thepatients.Such chronic inflammationinthevesselwallmayalsobeassociatedwithinfections andwith autoimmune diseasesthusindicating the involvement of both humoraland cell-mediatedimmunemechanismsinCHD. [35-38]

Neumannetaldidastudytodetermine theprognosticsignificanceofalteredplasmaviscosity andEAin unstableangina.Itwasastudy of96patientswithunstableangina.

Thepatientswere givenstandardizedprotocolmanagementandwereeitherfollowedupforsixmonths oruntilsurgicalintervention.Resultsshowedthatplasmaviscosity andEAwerebetterpredictorfor AMI thanage,gender,fibrinogenlevels,ST segmentchanges.Also,HoltermonitoringwithSTsegmentanalysisshowedthe ischemic episodes weremore common in cases with high EA. [39]

InastudybyRuhenstroth-Bamu,EAvaluewasstudied in groups of patients with and without CHD and in normal subjects using theMetricellapparatus.TheresultsshowedthatEAwasstrongly associated with MI and arteriosclerosis ofperipheralarteries. [40]

ArbeletalperformedastudyshowingEAasacauseofsluggishblood flow in ACS. [41]In1988,it was recommended by InternationalCommittee for StandardizationinHematology thatErythrocyte sedimentation ratecanbe a vitaltestfor the monitoringof chronic inflammatory processes. [42]Variousstudieswere done andErythrocyte sedimentation ratewasfoundtohave potential predictiveimportanceinCAD. [19,43]

ErikIngelssonetal found that Erythrocyte sedimentation rate couldbeastrong predictorofCADmortality.The study was performed on2,314 middle-agedmenfree fromHF,MI andvalvular heart disease.Infollow-uptimeof30years,282men developed HF.Thestudy concluded thatErythrocyte sedimentation ratewasa significantpredictor

ofHF,independentof other risk factorsfor HF andAMI.However,inthisstudy,Erythrocyte sedimentation ratewasdeterminedbyWestergren's method and Erythrocyte sedimentation rate greaterthan just 6 mm/hrwas considered to beapositivetest. [44]

Inanotherstudy by Erikssenetal,theErythrocyte sedimentation ratewasdeterminedin2014healthy menaged40–60yearsandthetestwasrepeated7yearslater.Erythrocyte sedimentation ratestrongly correlated withage,haemoglobinlevel,smoking status,total cholesterollevelandsystolicblood pressure.Erythrocyte sedimentation ratewas estimated usingWestergren'smethod.ESR>15mm/hr wasobservedinmenwhohaddevelopedanginaand/or hada positive stressElectrocardiogram test atthesecondsurvey.Thestudyconcluded by stating that Erythrocyte sedimentation rateisastrong predictorofCHD mortality. [45]

AndreaNatalietalalso performedastudy toevaluatetherelationbetweenErythrocyte sedimentation rateand theextensionofatherosclerosis(ATS)andCHDmortality.Itwasaretrospective study withfollowup periodof92months.Study was performedin1726patientsundergoing angiographyandsubsequentmortality wererelatedtoErythrocyte sedimentation rateandtootheriskfactors. Erythrocyte sedimentation ratewasprogressively higherinthepresenceof1,2,or3-vesseldiseaseand*Pvalue was*<0.0001.In

thefollow-upperiod,170patientsdiedofacardiaccause.Erythrocyte sedimentation ratewas consideredhighwhenitwas >18mmand>23 mm/1sthrin malesand femalesrespectively.ThestudyconcludedthatErythrocyte sedimentation rateisanindependentcorrelateofcoronaryATSand that itisa predictorofpoor prognosis inIHD. [46]

InthestudybyAtlaBhagyaLakshmietal,themeanErythrocyte sedimentation rateofASTEMI(29±17.34) was significantly higher than the mean Erythrocyte sedimentation rate of control group (15.5 ±12.37).PrognosisofthepatientswithASTEMIandcomparisonofgradeswith Erythrocyte sedimentation rate was not mentioned. [9]

JosephYayanalso performed astudyonpatientswithanginapectorisandmyocardial infarctionundergoingaheart catheterexaminationvscontrolgroup.The Erythrocyte sedimentation rate wasprolongedin79(58.09%)of 136patients.69(50.74%)patientshadCHD with prolongedErythrocyte sedimentation rate.The Erythrocyte sedimentation ratewasalsoprolongedinten(7.35%)patientswithoutCHD. ThespecificityoftheErythrocyte sedimentation rateforCHDwas70.59%andthesensitivitywas67.65% whichshowsthatErythrocyte sedimentation rate isnotalwaysadependablemarker.Thestudy concludedthat Erythrocyte

sedimentation ratemay beausefuladditionaldiagnosticcriterionforCHD. [47]Buttheassociation was termedas frequent ratherthan always.

Inviewofvarioussuchstudiesdone,ofalltheparametersavailable,Erythrocyte sedimentation ratecame out as a cheap, fast and widely applicable inflammatorymarker to assess inflammationandEAinpatientswithCAD.ButErythrocyte sedimentation ratecorrelates poorly withEAduetotheconfounding effectsof temperature,PCV, albumin,hemodilutionby anticoagulants.Erythrocyte sedimentation ratealsodoesnot differentiatewhetherEAcanbe a causeofcellularfactorsorof plasma factors. [29,33]The Erythrocyte sedimentation rateisa usefultestinclinicalpracticealsoasanindicator of otherinflammatoryprocesses,infection,traumaormalignantdisorders,stroke etc. As theageofpatientspronetocoronaryarterydiseasesandotherinflammatory processes likearthritis,vasculitisetc.iscommon;specificity ofthetestbecomes low.Andthisarise theneed ofatest that correlatesbetterto assess EA in CHD patients. [9,48]

Hysietalalsodidastudy proposingtheuseofphotoacoustics(PA)forthe detectionandcharacterizationof RBC aggregationvia 2D simulation. Sampleswere exposedto1064nmlaserirradiation. Various concentrationsofDextran-70 were usedtoinduce RBCs aggregation.Experimentalresultsconfirmedthat PA hadthe abilityto detect

and quantifyRBCs aggregation. [50]

Berlinerforthefirsttimedidastudy knownasslidetestontwothousandfive hundredeighty-sixindividuals.They excludedindividualswithchronicinflammatory diseases, collagenvascular disease, autoimmune disease or chronic skin disease. Bloodcount,Erythrocyte sedimentation rateand fibrinogenlevelswere estimated.Three volumesof venous bloodwastakenfollowingovernightfastwith1volumeof3.8%sodiumcitrate. One dropofthismixturewastrickledontheslideatanangleof30^{o}leavingafinefilm.Theslidesweredriedroomtemperature,stainedandscannedbyusinganimage analysis system (INFLAMET). [9,48]

156individualswerefoundto havevasculardisease. There were95menand61women(M:Fratioas3.1:2)atameanageof61.6+_13.6yrs. Thestudy wasnot performedonpatientswith a specificdiagnosisbutstillshowedthat EAmay beausefulbiomarkershowingsuperiorityoverother commonlyused markersto detectinflammation in subjects with atherosclerotic heart disease. [51]

AtlaBhagyaLakshmietal performedastudy withtheaimofusing aslide testtotestfor EAinsubjectswithacute STEMI, acuteischemicstrokeand healthycontrols and also to studythe prognostic

value of Erythrocyte sedimentation rateandEAATin predictingthe1-weekoutcomeofthesubjectsofacuteSTEMI andacuteischemic stroke.Theystudiedthirtysubjectsofacute STEMIwhowereadmittedwithin 6hrs of onset of pain in the chest,subjectswith pain lasting more than 30minutes and subjects with1mm STsegmentelevationinatleasttwocontiguouselectrocardiogram leads. [9]

EAcanbedeterminedaccurately by commercially available systems,the MyreneRheometerand cell flow meter. Itanalyzestheaggregatesizedistribution which is expressed asthenumberofcellsperaggregate,asafunctionofshearstress.However,they are incredibly expensiveandneeda high-end lab set-up. A simpleslidetechniqueusing citratedbloodwithimageanalysiswas designed by Israeli researchers to assess EA.

Zeltseretaldidasimilarstudy wheretheyadaptedasimilar technique torevealthestateofEAin 206patientswith ischemicheartdiseaseand134patientswithischemic neurological event. [52]

AtlaBhagyaLakshmietaltooka single largedropof citratedblood(one volumeof0.8%citratetothreevolumesofblood)andtrickledonaslidepositione dat 45°.Theslidewasleftinclinedfor10sec.leaving afinefilm. A similar procedure was followed by us. Adropofbloodwascollectedbeforecommencingany treatmenttothe patient. [9]

The slideswere stainedwithfour to five dropsofLeishmanstainandleft for two min. This was followedby twicetheamountof DW.After8min,theslideswere washed, dried and examined.The subjectswere on follow upfor a weekandtheoutcomes wererecorded. We deployed asimilar methodology in our study.Bloodsamplewascollected fromantecubitalveinimmediately afteradmission.Subsequently,thepatientswere managedaccording tothestandardtreatmentprotocolofourhospital.Erythrocyte sedimentation rateby Westergren'smethodandslide test,EAATwereperformedalongwithother investigations· [9]

Zeltseretaltookthree volumesofvenousbloodwithonevolumeof3.8% sodiumcitrate.Largedropsofthissamplewereplacedonaslidethatwasheld for two to three secondsonly at45°leavingafinefilm.Theslideswerethendriedandautomatic stainingwas performed bymeans of the HEMA TEK slide stainer. [52]

In thestudybyAtlaBhagyaLakshmi et al, among thirtypatients ofAMI,21weremalesand9werefemalesofagerangingfrom26to 80years.Maletofemaleratiowas2.3:1andthemeanageofthesubjects was51.87± 13.11years.Among 30subjects,therewereseventeenmalesandthirteenfemales. The sexratiowas1.3:1withmeanage of the controls, 40.966± 12.73 years.Zeltseretalexaminedatotalof134patientswithischemicheartdisease;89

males and 45 females, M:F ratio 1.99:1. [9]

Inourstudy,outof50patients,38weremalesand12werefemales,M:F ratio 3.1:1.The age of females ranged from 44-75 years, the average age being 60.5±10.55years.Theageofmalesrangedfrom28-77years,theaverageagebeing 55.5±10.5years.Combining themalesandfemales,the agerangedfrom 26-86years with the averageagebeing58.1±10.9years.

Inthestudy byAtlaBhagyaLakshmietal,50%(15of30)ofpatientsofAMI belongedtoCandDgrades.Amongthecontrols,only2ofthe30subjectsbelonged togradesCandD.Inourstudy,8patientsshowedGradeA,53patientsshowGrade B, 34 patients showedGradeC and 5 patients showed GradeDRBC aggregation. [9]

Inthestudy byZeltseretalEAwasnotassessedasgrades,rathercomparison wasmade onthebasisof variablescalledErythrocyte Percentage(*EP*),Aggregation Radius(*AR*)& VacuumRadius(*VR*).Asignificant(P<0.0001)correlationwasfound betweentheEAAandbothfibrinogenconcentrationsandESR.Importantly,there wasa subgroupof 49patientswhohadnormalfibrinogenconcentrations

anda highly significant increment in theEA. [52]

In our study, Outof50patients, 5 showedGradeARBC aggregationandallofthemhad good prognosis.

Nineteen 19 patients showed Grade B RBC aggregation, out of which 17 patients (89.5.6%) had good prognosis, one patient had arrhythmia and 1 patients died. Overall therewere2 patients with bad prognosis (10.5%).

Twenty one (21)showedGrade CRBC aggregation,outof which14 patients (66.7%) had good prognosis, 7 patients (33.3%) with bad prognosis.

Five (5)patientsshowedGradeDRBCaggregation.Only one(20%)hadgood prognosis.2patients(40%)hadarrhythmia and shock and2patients(40%)died.Overall4 patients (80%) had bad prognosis.

CorrelationofgradesofRBCaggregationwasdone withprognosisofthe patient and the result was statisticallysignificantwith p value0.006.

Conclusion

CONCLUSION

EAAT is a non-complex slide test performed in bedside. Theslides can beassessed subjectivelyandcan begradedbasedonthedegreeofaggregation. EA isa useful biomarkertodetectinternalinflammationinindividualswith athero-thrombotic risk factors.

The test mayhave the potential to assess the risk of ASTEMI andalsotoassessprognosisinsubjects withASTEMI.Inthe presentstudy,greaterEA wasseeninsubjects of ASTEMI andsignificantcorrelationwas found betweengrades ofEAand prognosis ofthepatient. Patients with severe grades of EA C&D are high risk individuals for complications. The high risk patients are candidates for PCI (delayed PCI).

Ideallyquantificationofaggregationshould alsoconsider the thicknessof the aggregate. Specificguidelines are tobe laiddownfor preparationofslides whichexplain the amountofbloodtobetaken,amountofanticoagulant tobe added,concentrationof the anticoagulant,angleof inclinationof slide andtime span ofinclination.

Inthe currentstudy,stainedslideswereassessedandgraded. They werefound tobeexcellentprognostic indicatorsinpatientswith MI.Itisasimplebedsideprocedure and is less expensive. Thus,it can beused asascreeningtestforhigh-risk individuals.

Therefore this study concludesthatslide testshowsbetter correlationwiththe prognosis andaidedwell to detect thepresenceofinternal inflammation in Acute STEMI.

Summary

SUMMARY

Erythrocyte adhesiveness and aggregation test isa usefulbiomarker todetectinternal inflammation in individuals with atherothromboticrisk factors. Atherosclerosis is associatedwithchronicinflammationofthearterialwall;therefore,many researchers have performed various studies overinflammatory markers and markers of erythrocyteadhesiveness and aggregationsoastofindreliableindicatorsofcoronary heart disease(CHD)risk and progression. [16,17,18,63]

EAcanbedeterminedaccurately bycommercially availablesystems.However,they are very expensive.ESRisacheap,fastandwidely applicableinflammatorymarkertoassessinflammationandEA inpatientswithCHD,butESRcorrelatespoorlywithEA dueto theconfounding effects. [64]

Sowedidastudy withtheaimofusing thesimpleslidetesttotestfor EAinsubjectswithacute ST-elevatedMI (ASTEMI)andtostudy theprognosticvalueofESRandEA and adhesivenesstest(EAAT)inpredicting the1-weekoutcomeofthese subjects.

Inthe presentstudy,stainedslideswereassessedandgraded.Greater EAwasseeninsubjectsof acute MIand

significantcorrelationwasfoundbetweengradesof EAand prognosisofthepatient.

Patients with severe grades of EA C&D are high risk individuals for complications. The high risk individuals are candidates for percutaneous intervension (delayed PCI) and hence, referred to higher centre for the same.

Inthepresentstudy,stainedslideswereassessedandgradedandwerefoun d tobegoodprognostic indicatorsinpatientswith MI.Itisasimplebedsideprocedure and is cost-effective. Thus,it can beused asascreeningtestforhigh-risk individuals.

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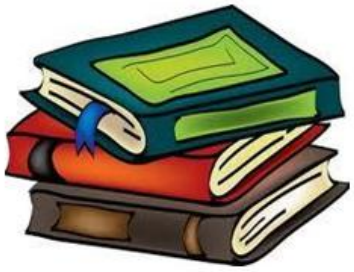

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Annexure-I

Proforma

PROFORMA

I.CASE NO:

II.TITLE:

Astudy toassessgradesofEAinacuteSTEMI and its prognostic significance.

III.SUBJECT INFORMATION:

Name:

Age:

Sex:

Address:

Occupation:

IV.PRESENT HISTORY:

COMPLAINTS:

- Pain in the cental or lateral chest wall (feeling oftightness or heaviness,pressure, orsqueezing)
- Pain radiating to lowerjaw, neck, left arm, back, orepigastric region
- Breathlessness or dyspnoe)
- Excessivesweating
- Palpitations
- Nausea,Vomiting
- LOC(loss of consciousness)

V.PAST HISTORY

- Any past historyofmedication
- DM/SHT
- Previous history heart Surgeries
- Hypercholestrolemiaor familial disorder

VI.PERSONAL HISTORY

- Diet:Vegetarian/ Non vegetarian
- Bladder and bowel habits:Regular/Irregular
- Addiction- chronic tobacco use, ethanol consumption, history of Smoking, drug abuse

VII.GENERAL EXAMINATION

- Built
- Nourishment
- Cyanosis
- Pallor
- Jaundice
- Pedal edema
- Lymphadenopathy

VIII.VITALS

1. Pulse: per min
   Rate, Rhythm, Character, Volume, Condition of the vessel wall, radio femoral delay
2. Blood pressure: mm/Hg
3. Temperature: °F
4. Respiratory Rate:

VIII.SYSTEM EXAMINATION

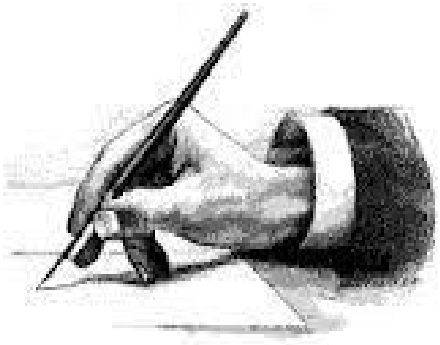
1. Cardiovascularsystem examination:
2. Respiratorysystem examination:
3. Abdomen:
4. Central nervous system examination:

IX. INVESTIGATIONS

1. EA AND ADHESIVENESS TEST
2. ECG:
3. COMPLETE BLOOD COUNT-TC,DC,HB
4. ERYTHROCYTE SEDIMENTAION RATE
5. RFT WITH ELECTROLYTES
6. LFT-ENZYMES,SGOT
7. LIPID PROFILE-TG,LDL,HDL,TOTAL CHOLESTEROL
8. CARDIAC ENZYMES – CPK, LDH
9. ECHO

Annexure–II

InformedConsent



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Annexure–III

Master Chart

MASTER CHART

Case No.	Name	Age	Sex	ECG	Erythro-cyte ag-gregation grade	ESR mm/ hr	LEUCO-CYTE COUNT Cells/cum m	ECHO	SGOT IU/L	Cardiac En-zymes		LIPID PROFILE				Prognos-sis with-in 1 Week period	Complications
										CPK-MB IU/L	TROP-T ng/L	TG mg%	LDL mg %	HDL mg %	TOTAL CHOLE-STEROL mg%		
1.	Ramdev	65	M	AW-MI	**B**	30	12100	RWMA AW	75	63	6.0	156	105	48	184	GOOD	NIL
2.	Srinivasan	76	M	IWMI	**B**	32	11600	RWMA IW	48	55	6.9	137	90	50	213	GOOD	NIL
3.	Nagavalli	60	F	IWMI	**B**	26	10300	RWMA IW	60	73	5.7	158	94	36	206	GOOD	NIL
4.	Rajeshwari	50	F	IWMI	**D**	30	11200	RWMA IW	72	80	7.1	166	86	42	190	BAD	DIED
5.	Kuppan	51	M	ASMI	**C**	24	10800	RWMA IVS	40	59	6.5	175	84	38	208	GOOD	NIL
6.	Raja	35	M	IWMI	**C**	20	9600	RWMA IW	65	79	7.0	159	110	41	200	BAD	ARRYTHMIA
7.	Ramesh	28	M	IWMI	**A**	22	11000	RWMA IW	55	67	6.3	142	120	40	190	GOOD	NIL
8.	Subramani	60	M	RVMI	**B**	26	11500	RWMA IW+P	35	51	5.4	168	115	32	208	GOOD	NIL
9.	Madhan	45	M	ALMI	**C**	20	10400	RWMA AW	40	55	5.9	172	95	45	220	GOOD	NIL
10.	Saravanan	45	M	AW-MI	**A**	28	12200	RWMA AW	45	60	7.3	160	98	37	189	GOOD	NIL
11.	Kowsalya	75	F	AW-MI	**B**	30	13000	RWMA AW	54	70	6.2	175	89	43	225	GOOD	NIL
12.	Baskar	45	M	IPWMI	**B**	26	10300	RWMA IW+P	70	81	6.3	166	85	50	230	GOOD	NIL
13.	Moideenkhan	56	M	IWMI	**C**	25	9800	RWMA IW	62	50	6.8	109	118	42	180	GOOD	NIL
14.	Venkat	53	M	IWMI	**A**	28	9700	RWMA IW	72	52	6.0	173	95	37	210	GOOD	NIL
15.	Sekar	58	M	IPWMI	**B**	30	8600	RWMA IW+P	85	48	7.3	153	99	39	189	GOOD	NIL
16.	Edward	32	M	EXT AW-	**C**	20	11000	RWMA AW	64	58	6.5	148	115	33	186	GOOD	NIL

Case No.	Name	Age	Sex	ECG	Erythro-cyte ag-gregation grade	ESR mm/ hr	LEUCO-CYTE COUNT Cells/cum m	ECHO	SGOT IU/L	Cardiac En-zymes		LIPID PROFILE				Prognos-sis with-in 1 Week period	Complications
										CPK-MB IU/L	TROP-T ng/L	TG mg%	LDL mg %	HDL mg %	TOTAL CHOLE-STEROL mg%		
				MI													
17.	Chinnathai	64	F	AW-MI	**D**	32	9400	RWMA AW	58	62	5.5	168	86	47	205	BAD	DIED
18.	Ramesh	38	M	IWMI	**C**	18	12200	RWMA IW	45	66	6.7	156	100	50	176	GOOD	NIL
19.	Sumathi	65	F	IWMI	**C**	28	11500	RWMA IW	60	53	6.2	175	105	40	212	BAD	DIED
20.	Bakyaraj	57	M	IWMI	**B**	24	7900	RWMA IW	75	69	5.3	167	112	36	209	GOOD	NIL
21.	Mamtha	75	F	IPW MI	**C**	28	9200	RWMA IW+P	95	65	5.9	162	92	34	216	BAD	DIED
22.	Ravi	46	M	IWMI	**C**	22	8900	RWMA IW	80	72	7.0	153	96	38	198	GOOD	NIL
23.	Mangalaksmi	77	F	AW-MI	**B**	24	10200	RWMA AW	70	50	6.6	150	85	30	188	GOOD	NIL
24.	Thiruvengadam	57	M	IWMI	**C**	28	11300	RWMA IW	48	57	5.7	148	117	44	192	GOOD	NIL
25.	Lakshmi	61	F	AW-MI	**C**	30	10700	RWMA AW	68	49	6.4	166	110	46	202	BAD	ARRYTHMIA
26.	Rajamuthu	41	M	AW-MI	**A**	20	11800	RWMA AW	52	71	6.9	154	106	31	190	GOOD	NIL
27.	Velayutham	47	M	EXT AW-MI	**C**	24	12000	RWMAAW	85	76	5.3	164	112	48	212	BAD	DIED
28.	Muthu	50	M	IWMI	**B**	20	9600	RWMA IW	78	56	6.9	159	106	35	209	GOOD	NIL
29.	Louis	59	M	ALMI	**A**	20	11200	RWMA AW	55	63	7.3	149	98	39	185	GOOD	NIL
30.	Murugan	52	M	IWMI	**B**	28	10400	RWMA IW	60	71	5.9	132	92	40	179	GOOD	NIL
31.	Ibrahim	50	M	AW-MI	**D**	23	12200	RWMA IW	64	85	6.0	172	106	47	220	BAD	LVF
32.	Vijayan	44	M	IPW MI	**B**	19	9000	RWMA IW+P	90	81	5.7	154	86	36	201	GOOD	NIL
33.	Anthonyamma	70	F	AW-MI	**C**	30	10500	RWMA AW	52	63	7.0	169	120	40	216	GOOD	NIL

Case No.	Name	Age	Sex	ECG	Erythro-cyte ag-gregation grade	ESR mm/ hr	LEUCO-CYTE COUNT Cells/cum m	ECHO	SGOT IU/L	Cardiac En-zymes		LIPID PROFILE				Prognо-sis with-in 1 Week period	Complications
										CPK-MB IU/L	TROP-T ng/L	TG mg%	LDL mg %	HDL mg %	TOTAL CHOLE-STEROL mg%		
34.	Sivakumar	63	M	EXT AW-MI	**D**	32	11200	RWMA AW	65	58	5.5	170	82	33	207	BAD	ARRYTHMIA
35.	Laksmi	50	F	AW-MI	**B**	25	9000	RWMA AW	48	67	6.3	163	87	49	193	GOOD	NIL
36.	Venkatesan	30	M	EXT AW	**C**	18	9600	RWAM AW	52	77	7.4	173	94	50	208	BAD	ARRYTHMIA
37.	Basha	38	M	IWMI	**B**	20	8600	RWMA IW	80	69	6.7	165	108	42	220	GOOD	NIL
38.	Palani	60	M	IWMI	**C**	30	10200	RWMA IW	45	56	5.4	157	93	46	180	GOOD	NIL
39.	Ganesan	52	M	AW-MI	**B**	20	13200	RWMA AW	88	49	6.4	175	99	35	225	GOOD	NIL
40.	Egambaram	55	M	AW-MI	**C**	27	11400	RWMA AW	40	58	5.8	140	106	38	190	GOOD	NIL
41.	Subramani	39	M	IWMI	**B**	20	9800	RWMA IW	40	54	5.2	120	86	38	197	GOOD	NIL
42.	Fathima	44	F	IWMI	**B**	22	11000	RWMA IW	76	61	6.3	146	110	45	192	GOOD	NIL
43.	Salim	55	M	IWMI	**C**	27	10400	RWMA IW	45	62	7.1	153	90	38	212	BAD	CARDIOGENIC-SHOCK
44.	Syed	45	M	RVMI	**C**	28	12500	RWMA IW+P	82	71	6.6	160	100	40	206	GOOD	NIL
45.	Harinath	49	M	AW-MI	**B**	25	11600	RWMA AW	70	82	7.3	136	120	48	217	BAD	LVF
46.	Syedbasha	62	M	HLMI	**B**	30	12000	NO RWMA	80	73	6.7	170	96	40	230	BAD	DIED
47.	Kanniyappan	58	M	ASMI	**C**	28	11500	RWMA SEPT	56	67	6.1	145	88	44	182	GOOD	NIL
48.	Emmanuel	50	M	AW-MI	**C**	26	8800	RWMA AW	65	48	5.8	165	92	38	197	GOOD	NIL
49.	Ayusha	65	F	IPWMI	**C**	30	9200	RWMA IW+P	35	51	7.4	151	96	50	186	GOOD	NIL
50.	Mohan	60	M	IWMI	**D**	27	9000	RWMA IW	50	65	5.5	163	88	46	204	GOOD	NIL